



Regulatory Impact Analysis for the Final Clean Air Interstate Rule

Regulatory Impact Analysis for the Final Clean Air Interstate Rule

U.S. Environmental Protection Agency Office of Air and Radiation

Air Quality Strategies and Standards Division, Emission, Monitoring, and Analysis Division and Clean Air Markets Division

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CHAPTER 1

EXECUTIVE SUMMARY

This Regulatory Impact Analysis (RIA) presents the health and welfare benefits and the costs of the Clean Air Interstate Rule (CAIR) and compares the benefits to the costs of implementing CAIR in 2010 and 2015.

1.1 Results

Synopsis

EPA has estimated the benefits and costs of the Clean Air Interstate Rule and finds that the rule results in estimated annual net benefits of \$71.4 or \$60.4 in 2010 and \$98.5 or \$83.2 billion in 2015. These alternate net benefit estimates reflect differing assumptions about the social discount rate used to estimate the social benefits and costs of the rule. The lower estimates reflect a discount rate of 7 percent and the higher estimates a discount rate of 3 percent. In 2015, the total annual quantified benefits are \$101 or \$86.3 billion and the annual social costs are \$2.6 or \$3.1 billion—benefits outweigh social costs in 2015 by a ratio of 39 to 1 or 28 to 1 (3 percent and 7 percent discount rates respectively). An alternative comparison of the annual benefits of the rule to the estimated private costs to the electric generating industry in 2015 result in benefits outweighing costs by a ratio of 25 to 1 (benefits of \$101 billion compared to costs of \$3.6 billion). These estimates do not include the value of benefits or costs that we cannot monetize. Upon consideration of the uncertainties and limitations in the analysis, it remains clear that the benefits of CAIR are substantial and far outweigh the costs.

A comparison of the benefits and costs of the rule in 2010 and 2015 is shown in Table 1-1. The benefits and costs reported for CAIR in Table 1-1 represent estimates for a complete CAIR program that includes the CAIR promulgated rule and the concurrent proposal to include annual sulfur dioxide (SO₂) and nitrogen oxide (NO_x) controls for New Jersey and Delaware. The modeling used to provide these estimates also assumes annual SO₂ and NO_x controls for Arkansas that are not a part of the complete CAIR program resulting in a slight overstatement of the reported benefits and costs.

Description	2010	2015
Social costs ^b		
3 percent discount rate	\$1.91	\$2.56
7 percent discount rate	\$2.14	\$3.07
Social benefits ^{c,d,e}		
3 percent discount rate	73.3 + B	101 + B
7 percent discount rate	62.6 + B	86.3 + B
Health-related benefits:		
3 percent discount rate	72.1	99.3
7 percent discount rate	61.4	84.5
Visibility benefits	1.14	1.78
Net benefits (benefits-costs) ^{e,f}		
3 percent discount rate	\$71.4 + B	\$98.5 + B
7 percent discount rate	\$60.4 + B	\$83.2 + B

 Table 1-1. Summary of Annual Benefits, Costs, and Net Benefits of the Clean Air Interstate Rule^a (billions of 1999\$)

^a All estimates are rounded to three significant digits and represent annualized benefits and costs anticipated for the year 2010 and 2015. Estimates relate to the complete CAIR program including the CAIR promulgated rule and the proposal to include SO₂ and annual NO_x controls for New Jersey and Delaware. Modeling used to develop these estimates assumes annual SO₂ and NO_x controls for Arkansas resulting in a slight overstatement of the reported benefits and costs for the complete CAIR program.

- ^b Note that costs are the annualized total costs of reducing pollutants including NO_x and SO₂ for the EGU source category in the CAIR region.
- ^c As this table indicates, total benefits are driven primarily by PM-related health benefits. The reduction in premature fatalities each year accounts for over 90 percent of total monetized benefits in 2015. Benefit estimates in this table are nationwide (with the exception of ozone and visibility) and reflect NO_x and SO₂ reductions. The analysis assumes that States will choose to achieve CAIR caps solely from the EGU source category. Ozone benefits represent benefits in the eastern United States. Visibility benefits represent benefits in Class I areas in the southeastern United States.
- ^d Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Potential benefit categories that have not been quantified and monetized are listed in Table 1-4.
- ^e Valuation assumes discounting over the SAB-recommended 20-year segmented lag structure described in Chapter 4. Results reflect 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (U.S. EPA, 2000; OMB, 2003).
- ^f Net benefits are rounded to the nearest \$100 million. Columnar totals may not sum due to rounding.

Details of the important analysis assumptions, including entities regulated, baseline, analysis year, control scenario, and other relevant analysis assumptions are discussed in Chapter 2 of this report.

1.1.1 Health Benefits

CAIR is expected to yield significant health benefits by reducing emissions of two key contributors to fine particle and ozone formation. Sulfur dioxide contributes to the formation of fine particle pollution ($PM_{2.5}$), and nitrogen oxide contributes to the formation of both $PM_{2.5}$ and ground-level ozone.¹

Our analyses suggest CAIR would yield benefits in 2015 of \$101 billion (based on a 3 percent discount rate) and \$86.3 billion (based on a 7 percent discount rate) that includes the value of avoiding approximately 17,000 premature deaths, 22,000 nonfatal heart attacks, 12,300 hospitalizations for respiratory and cardiovascular diseases, 1.7 million lost work days, 500,000 school absences, and 10.6 million days when adults restrict normal activities because of respiratory symptoms exacerbated by $PM_{2.5}$ and ozone pollution.²

We also estimate substantial additional health improvements for children from reductions in upper and lower respiratory illnesses, acute bronchitis, and asthma attacks. See Table 1-2 for a list of the annual reduction in health effects expected in 2010 and 2015 and Table 1-3 for the estimated value of those reductions.

1.1.2 Welfare Benefits

The term *welfare benefits* covers both environmental and societal benefits of reducing pollution, such as reductions in damage to ecosystems, improved visibility and improvements in recreational and commercial fishing, agricultural yields, and forest

¹ Although well over 90 percent of the expected benefits of this rule are derived from reductions in SO_2 and NO_x , a small portion of EPA's projected benefits are a result of reductions in primary PM from power plants. Although this reduction is not required by the rule, it is a potential ancillary benefit of installing certain SO_2 control technologies.

² These estimates account for growth in the public's willingness to pay for reductions in health and environmental risks and account for growth in real gross domestic product per capita between the present and 2015. Benefit estimates reflect the use of 3 percent and 7 percent discount rates consistent with the U.S. Environmental Protection Agency (EPA) and the Office of Management and Budget (OMB) guidelines for preparing economic analyses (U.S. EPA, 2000; OMB, 2003).

Table 1-2. Clean Air Interstate Rule: Estimated Reduction in Incidence of Adverse Health Effects^a

	2010	2015
Health Effect	Incidence Reduction	
PM-Related Endpoints:		
Premature mortality ^{b,c}		
Adult, age 30 and over	13,000	17,000
Infant, age <1 year	29	36
Chronic bronchitis (adult, age 26 and over)	6,900	8,700
Non-fatal myocardial infarction (adults, age 18 and older)	17,000	22,000
Hospital admissions—respiratory (all ages) ^d	4,300	5,500
Hospital admissions—cardiovascular (adults, age >18) ^e	3,800	5,000
Emergency room visits for asthma (age 18 years and younger)	10,000	13,000
Acute bronchitis (children, age 8-12)	16,000	19,000
Lower respiratory symptoms (children, age 7-14)	190,000	230,000
Upper respiratory symptoms (asthmatic children, age 9–18)	150,000	180,000
Asthma exacerbation (asthmatic children, age 6-18)	240,000	290,000
Work loss days (adults, age 18-65)	1,400,000	1,700,000
Minor restricted-activity days (MRADs) (adults, age 18-65)	8,100,000	9,900,000
Ozone-Related Endpoints		
Hospital admissions—respiratory causes (adult, 65 and older) ^f	610	1,700
Hospital admissions—respiratory causes (children, under 2)	380	1,100
Emergency room visit for asthma (all ages)	100	280
Minor restricted-activity days (MRADs) (adults, age 18-65)	280,000	690,000
School absence days	180,000	510,000

Incidences are rounded to two significant digits. These estimates represent benefits from CAIR nationwide. The modeling used to derive these incidence estimates are reflective of those expected for the final CAIR program including the CAIR promulgated rule and the proposal to include SO_2 and annual NO_x controls for New Jersey and Delaware. Modeling used to develop these estimates assumes annual SO_2 and NO_x controls for Arkansas resulting in a slight overstatement of the reported benefits and costs for the complete CAIR program.

^b Premature mortality benefits associated with ozone are not analyzed in the primary analysis.

^c Adult premature mortality based upon studies by Pope et al., 2002. Infant premature mortality is based upon studies by Woodruff, Grillo, and Schoendorf, 1997.

^d Respiratory hospital admissions for PM include admissions for chronic obstructive pulmonary disease (COPD), pneumonia, and asthma.

^e Cardiovascular hospital admissions for PM include total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

^f Respiratory hospital admissions for ozone include admissions for all respiratory causes and subcategories for COPD and pneumonia.

Table 1-3. Estimated Monetary Value of Reductions in Incidence of Health and Welfare Effects (in millions of 1999\$)^{a,b}

		2010	2015
Health Effect	Pollutant	Estimated Value of Reductions	
Premature mortality ^{c,d} Adult >30 years		¢(7.200	¢0 2 000
3% discount rate 7% discount rate Child <1 year	PM _{2.5}	\$67,300 \$56,600 \$168	\$92,800 \$78,100 \$222
Chronic bronchitis (adults, 26 and over)	PM _{2.5}	\$2,520	\$3,340
Nonfatal acute myocardial infarctions 3% discount rate 7% discount rate	PM _{2.5}	\$1,420 \$1,370	\$1,850 \$1,790
Hospital admissions for respiratory causes	PM _{2.5} , O ₃	\$45.2	\$78.9
Hospital admissions for cardiovascular causes	PM _{2.5}	\$80.7	\$105
Emergency room visits for asthma	PM _{2.5} ,O ₃	\$2.84	\$3.56
Acute bronchitis (children, age 8-12)	PM _{2.5}	\$5.63	\$7.06
Lower respiratory symptoms (children, 7-14)	PM _{2.5}	\$2.98	\$3.74
Upper respiratory symptoms (asthma, 9-11)	PM _{2.5}	\$3.80	\$4.77
Asthma exacerbations	PM _{2.5}	\$10.3	\$12.7
Work loss days	PM _{2.5} ,	\$180	\$219
Minor restricted-activity days (MRADs)	PM _{2.5} ,O ₃	\$422	\$543
School absence days	O_3	\$12.9	\$36.4
Worker productivity (outdoor workers, 18–65)	O_3	\$7.66	\$19.9
Recreational visibility, 81 Class I areas	PM _{2.5}	\$1,140	\$1,780
Monetized Total ^e Base Estimate:			
3% discount rate 7% discount rate	PM _{2.5} ,O ₃	\$73,300 + B \$62,600 + B	\$101,000 + B \$86,300 + B

Monetary benefits are rounded to three significant digits. These estimates represent benefits from CAIR nationwide for NO_x and SO_2 emission reductions from electricity-generating units (EGU) sources (with the exception of ozone and visibility benefits). Ozone benefits relate to the eastern United States. Visibility benefits relate to Class I areas in the southeastern United States. The benefit estimates reflected relate to the final CAIR program that includes the CAIR promulgated rule and the proposal to include SO_2 and annual NO_x controls for New Jersey and Delaware. Modeling used to develop these estimates assumes annual SO_2 and NO_x controls for Arkansas resulting in a slight

overstatement of the reported benefits and costs for the complete CAIR program. Monetary benefits adjusted to account for growth in real GDP per capita between 1990 and the analysis year (2010 or 2015). с

2015).
Valuation assumes discounting over the SAB-recommended 20-year segmented lag structure described in Chapter 4.
Results show 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (U.S. EPA, 2000; OMB, 2003).
Adult premature mortality based upon studies by Pope et al., 2002. Infant premature mortality based upon studies by Woodruff, Grillo, and Schoendorf, 1997.
B represents the monetary value of health and welfare benefits not monetized. A detailed listing is provided in Table 1-4. d

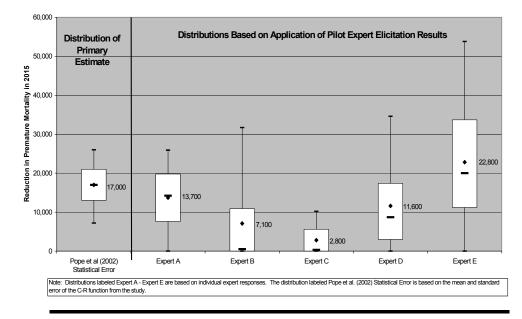
productivity. Although we are unable to monetize all welfare benefits, EPA estimates CAIR will yield welfare benefits of \$1.8 billion in 2015 (1999\$) for visibility improvements in southeastern Class I (national park) areas.

1.1.3 Uncertainty in the Benefits Estimates

As part of an overall program to improve the Agency's characterization of uncertainties in health benefits analyses, we present two types of probabilistic approaches to characterize uncertainty. The first approach generates a distribution of benefits based on the classical statistical error expressed in the underlying health effects and economic valuation studies used in the benefits modeling framework. The second approach uses the results from a pilot expert elicitation project designed to characterize key aspects of uncertainty in the ambient $PM_{2.5}$ /mortality relationship, and augments the uncertainties in the mortality estimate with the statistical error reported for other endpoints in the benefit analysis. Both approaches provide insights into the likelihood of different outcomes and about the state of knowledge regarding the benefits estimates.

The uncertainty estimates have the strength of presenting a statistical measure of the uncertainty in the underlying studies serving as the basis for the estimates used in the analysis. However, this approach captures only a limited portion of the uncertainty about the parameters. The 5th and 95th percentile points of the distributions are based on statistical error and cross-study variability and provide some insight into how uncertain our estimate is with regard to those sources of uncertainty. However, it does not capture other sources of uncertainty regarding the model specification and other inputs to the model, including emissions, air quality, and aspects of the health science not captured in the studies, such as the likelihood that PM is causally related to premature mortality and other serious health effects.

Figure 1-1 presents box plots of the distributions of the reduction in $PM_{2.5}$ -related premature mortality based on the C-R distributions provided by each expert, as well as that for our primary estimate, based on the statistical error associated with Pope et al. (2002). The distributions are depicted as box plots with the diamond symbol (\blacklozenge) showing the mean, the dash (–) showing the median (50th percentile), the box defining the interquartile range (bounded by the 25th and 75th percentiles), and the whiskers defining the 90 percent confidence interval (bounded by the 5th and 95th percentiles of the distribution). Our primary estimate based on the Pope et al. (2002) study shows that the average number of premature deaths avoided in 2015 is 17,000 (at a value of \$100 billion) with the 5th and 95th percentiles of the distribution ranging from 6,000 to 27,000 fewer mortalities. The figure



Note: The results of the Pilot Expert Elicitation are presented here as an illustration of EPA's initial efforts to characterize the uncertainties associated with the estimate of benefits from the $PM_{2.5}$ /mortality relationship. The Pilot was limited in scope and does not address inherent differences in the thought processes and background information used by each expert to express their distribution. Based on findings from the Pilot and a favorable peer review of the Pilot, EPA is conducting a full-scale expert elicitation to better characterize uncertainty in the mortality estimate for future regulatory analyses. See Appendix B for a full description of the two approaches used to characterize uncertainty.

Figure 1-1. Comparative Assessment of Relative Range of Uncertainty in Estimated Avoided Incidence of Premature Mortality Using Classical Statistical Error and the Pilot Expert-Based Characterizations of Uncertainty

shows that the average annual number of premature deaths avoided in 2015 based on the pilot expert elicitation ranges from approximately 3,000 (based on the judgments of Expert C), which is valued at \$23 billion to 23,000 (based on the judgments of Expert E), which is valued at \$140 billion. The confidence intervals vary across experts with all experts estimating zero at the 5th percentile and the 95th percentile ranging from 10,000 to 54,000 fewer mortalities.

As part of the CAIR analysis, we conducted a variety of supplemental analyses designed to provide the reader with an understanding of the degree of uncertainty that may be associated with the benefits resulting from implementation of this regulation. Because estimates of premature mortality contribute the most to the monetized benefits, our efforts focused on the sensitivity of the final benefits estimate to analytic judgments regarding this relationship. Specifically, we conducted analyses designed to characterize the degree of uncertainty in the slope (magnitude) of the $PM_{2.5}$ concentration-response function, the form of the $PM_{2.5}$ concentration-response function (i.e., the potential for a threshold), and the cessation lag (i.e., temporal relationship between cessation of exposure and reduction in adverse health effects). Both discrete and probabilistic approaches were used to characterize the uncertainty associated with the concentration response function.

These supplemental analyses yield the following insights:

- Use of statistical error associated with the ACS (Pope et al., 2002) estimate for the concentration response function for $PM_{2.5}$ —premature mortality as well as the statistical error associated with the concentration response functions for each of the other health endpoints to describe the probability distribution of total benefits yields a distribution in which the 95th percentile is nearly twice the mean (\$100 billion in 2015) and 5th percentile is one fourth the mean. The overall range from 5th to 95th percentile on the total benefits estimate represents one order of magnitude (\$26 billion to \$210 billion).
- Description of the probability distribution of the concentration response function for $PM_{2.5}$ —premature mortality using the results from the pilot expert elicitation (rather than the estimate based on the statistical error associated with the ACS cohort) yields a larger degree of uncertainty because the elicitation exercise was designed to encompass a broader set of model uncertainty. The mean annual benefits for each expert elicited during the pilot project range from approximately \$16 billion to \$130 billion in 2015
- Substitution of the steeper concentration response function for $PM_{2.5}$ —premature mortality from the Six Cities study increases the value of the total benefits from \$101 billion to \$208 billion in 2015.
- Substitution of the most plausible alternative lag structures has little overall impact on the estimate of total benefits (reductions are on the order of 5 to 15 percent).
- The assessment of alternative assumptions regarding the existence (and level) of a threshold in the $PM_{2.5}$ premature mortality concentration response function highlights the sensitivity of the analysis to this assumption. Only 5percent of the estimated premature mortality is due to changes in exposure above 15mg/m^3 , while over 84 percent of the premature morality related benefits are due to changes in PM_{2.5} concentrations occurring above 10ug/m^3 .

• Estimates of premature mortality from ozone exposure may result in an additional 500 premature deaths avoided and an increase in the estimated health benefits of CAIR by approximately \$3 billion annually.

In addition to these mortality related supplemental analyses, we also conducted analyses related to non-health (welfare) effects, including visibility and household cleaning costs. Based on these analyses, expanded coverage of welfare effects could increase benefits by over \$500 million. Other welfare effects have been quantified such as nitrogen and sulfur deposition reductions in the CAIR region, acidification reductions in lakes the Adirondacks and the northeastern US, and reduced nitrogen deposition to the Chesapeake Bay. While monetized estimates of these benefits could not be examined even in sensitivity analyses, it is likely these benefit categories are significant in terms of the total ecological endpoints.

1.2 Not All Benefits Quantified

EPA was unable to quantify or monetize all of the health and environmental benefits associated with CAIR. EPA believes these unquantified benefits are substantial, including the value of increased agricultural crop and commercial forest yields, visibility improvements, reductions in nitrogen and acid deposition and the resulting changes in ecosystem functions, and health and welfare benefits associated with reduced mercury emissions. Table 1-4 provides a list of these benefits.

1.3 Costs and Economic Impacts

For the affected region, the projected annual incremental private costs of CAIR to the power industry are \$2.36 billion in 2010 and \$3.57 billion in 2015. These costs represent the total cost to the electricity-generating industry of reducing NO_x and SO_2 emissions to meet the caps set out in the rule. Estimates are in 1999 dollars. Costs of the rule are estimated using the Integrated Planning Model and assume firms make decisions using costs of capital ranging from 5.34 percent to 6.74 percent.

In estimating the net benefits of regulation, the appropriate cost measure is "social costs." Social costs represent the welfare costs of the rule to society. These costs do not consider transfer payments (such as taxes) that are simply redistributions of wealth. The social costs of this rule are estimated to be \$1.91 billion in 2010 and \$2.56 billion in 2015 assuming a 3 percent discount rate. These costs become \$2.14 billion in 2010 and \$3.07 billion in 2015, if one assumes a 7 percent discount rate.

Pollutant/Effect	Effects Not Included in Primary Estimates—Changes in:
Ozone—Health ^a	 Premature mortality^b Chronic respiratory damage Premature aging of the lungs Nonasthma respiratory emergency room visits Increased exposure to UVb
Ozone—Welfare	 Yields for: commercial forests, fruits and vegetables, and commercial and noncommercial crops Damage to urban ornamental plants Recreational demand from damaged forest aesthetics Ecosystem functions Increased exposure to UVb
PM—Health ^c	 Premature mortality: short-term exposures^d Low birth weight Pulmonary function Chronic respiratory diseases other than chronic bronchitis Nonasthma respiratory emergency room visits Exposure to UVb (+/-)^e
PM—Welfare	 Visibility in many Class I areas Residential and recreational visibility in non-Class I areas Soiling and materials damage Ecosystem functions Exposure to UVb (+/-)^e
Nitrogen and Sulfate Deposition—Welfare	 Commercial forests due to acidic sulfate and nitrate deposition Commercial freshwater fishing due to acidic deposition Recreation in terrestrial ecosystems due to acidic deposition Existence values for currently healthy ecosystems Commercial fishing, agriculture, and forests due to nitrogen deposition Recreation in estuarine ecosystems due to nitrogen deposition Ecosystem functions Passive fertilization due to nitrogen deposition
Mercury Health	 Incidence of neurological disorders Incidence of learning disabilities Incidence of developmental delays Potential reproductive effects^f Potential cardiovascular effects^f, including: Altered blood pressure regulation^f Increased heart rate variability^f Incidence of myocardial infarction^f
Mercury Deposition Welfare	 Impacts on birds and mammals (e.g., reproductive effects) Impacts to commercial, subsistence, and recreational fishing

 Table 1-4.
 Unquantified and Nonmonetized Effects of the Clean Air Interstate Rule

Table 1-4.Unquantified and Nonmonetized Effects of the Clean Air Interstate Rule
(continued)

- ^a In addition to primary economic endpoints, there are a number of biological responses that have been associated with ozone health effects including increased airway responsiveness to stimuli, inflammation in the lung, acute inflammation and respiratory cell damage, and increased susceptibility to respiratory infection. The public health impact of these biological responses may be partly represented by our quantified endpoints.
- ^b Premature mortality associated with ozone is not currently included in the primary analysis. Recent evidence suggests that short-term exposures to ozone may have a significant effect on daily mortality rates, independent of exposure to PM. EPA is currently conducting a series of meta-analyses of the ozone mortality epidemiology literature. EPA will consider including ozone mortality in primary benefits analyses once a peer-reviewed methodology is available.
- ^c In addition to primary economic endpoints, there are a number of biological responses that have been associated with PM health effects including morphological changes and altered host defense mechanisms. The public health impact of these biological responses may be partly represented by our quantified endpoints.
- ^d While some of the effects of short term exposures are likely to be captured in the estimates, there may be premature mortality due to short term exposure to PM not captured in the cohort study upon which the primary analysis is based.
- ^e May result in benefits or disbenefits. See discussion in Section 5.3.4 for more details.
- ^f These are potential effects as the literature is insufficient.

Retail electricity prices are projected to increase roughly 2.0 to 2.7 percent with CAIR in the 2010 and 2015 time frame and then drop below 2.0 percent thereafter. The effects of CAIR on natural gas prices and the power-sector generation mix is also small, with a 1.6 percent or less increase in gas prices projected from 2010 to 2020. There will be a continued reliance on coal-fired generation, which is projected to remain at roughly 50 percent of total electricity generated. A relatively small amount of coal-fired capacity, about 5.3 GW (1.7 percent of all coal-fired capacity and 0.5 percent of all generating capacity), is projected to be uneconomic to maintain. In practice units projected to be uneconomic to maintain may be "mothballed," retired, or kept in service to ensure transmission reliability in certain parts of the grid. For the most part, these units are small and infrequently used generating units that are dispersed throughout the CAIR region. As demand grows in the future, additional coal-fired generation is projected to be built under CAIR and utilization of coal-fired units will increase. Because of this, coal production is projected to increase from 2003 levels by about 15 percent in 2010 and by 25 percent by 2020, and we expect greater coal production in Appalachia and the Interior coal regions of the country with CAIR. Overall, the impacts of CAIR are modest, particularly in light of the large projected benefits of CAIR.

1.4 Limitations

Every analysis examining the potential benefits and costs of a change in environmental protection requirements is limited to some extent by data gaps, limitations in model capabilities (such as geographic coverage), and variability or uncertainties in the underlying scientific and economic studies used to configure the benefit and cost models. Despite these uncertainties, we believe this benefit-cost analysis provides a reasonable indication of the expected economic benefits and costs of CAIR in future years.

For this analysis, such uncertainties include possible errors in measurement and projection for variables such as population growth and baseline incidence rates; uncertainties associated with estimates of future-year emissions inventories and air quality; variability in the estimated relationships between changes in pollutant concentrations and the resulting changes in health and welfare effects; and uncertainties in exposure estimation. We have used sensitivity analyses to address these limitations where possible.

EPA's cost estimates assume that all States in the CAIR region fully participate in the cap and trade programs that reduce SO_2 and NO_x emissions from EGUs. The cost projections also do not take into account the potential for advancements in the capabilities of pollution control technologies for SO_2 and NO_x removal and other compliance strategies, such as fuel switching or the reductions in their costs over time. EPA projections also do not take into account demand response (i.e., consumer reaction to electricity prices) because the consumer response is likely to be relatively small, but the effect on lowering private compliance costs may be substantial. Costs may be understated since an optimization model was employed and the regulated community may not react in the same manner to comply with the rules. The Agency also did not factor in the costs and/or savings for the government to operate the CAIR program as opposed to other air pollution compliance programs and transactional costs and savings from CAIR's effects on the labor supply. A listing of possible unquantified costs associated with the CAIR program are shown in Table 1-5.

Table 1-5. Unquantified Costs of the Clean Air Interstate Rule

Effects	Not	Quantified
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Employment shifts as workers are retrained at the same company or re-employed elsewhere in the economy.

Costs of running and administering the program to State and Federal Government.

Certain relatively small permitting costs associated with Title IV that new program entrants face.

1.5 References

- Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- U.S. Environmental Protection Agency (EPA). September 2000. *Guidelines for Preparing Economic Analyses*. EPA 240-R-00-003.
- U.S. Office of Management and Budget (OMB). 2003. Circular A-4 Guidance to Federal Agencies on Preparation of Regulatory Analysis.
- Woodruff, T.J., J. Grillo, and K.C. Schoendorf. 1997. "The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Infant Mortality and Particulate Air Pollution in the United States." *Environmental Health Perspectives* 105(6):608-612.

CHAPTER 2

INTRODUCTION AND BACKGROUND

2.1 Introduction

For this rulemaking, the U.S. Environmental Protection Agency (EPA) has assessed the role that transported emissions from upwind states play in contributing to unhealthy levels of $PM_{2.5}$ and 8-hour ozone in downwind states. Based on this assessment, the Clean Air Interstate Rule (CAIR) requires air emissions reductions from upwind states. This document presents the health and welfare benefits of CAIR and compares the benefits of this rule to the estimated costs of implementing the rule in 2010 and 2015. This chapter contains background information relative to the rule and an outline of the chapters of the report.

2.2 Background

Congress recognized that interstate pollution transport from upwind states can contribute to unhealthy pollution levels in downwind states. Therefore, the Clean Air Act (CAA) contains provisions in Section 110(a)(2)(D) that require upwind states to eliminate emissions that contribute significantly to nonattainment downwind. Under Section 110(a)(2), states are required to submit plans to EPA within 3 years of issuance of a revised National Ambient Air Quality Standard (NAAQS). Among other requirements, these plans are required to prohibit emissions in the state that contribute significantly to nonattainment downwind.

EPA's final rule finds that 28 states and the District of Columbia contribute significantly to nonattainment, or interfere with maintenance, of the NAAQS for $PM_{2.5}$ and/or 8-hour ozone in downwind states. EPA requires these upwind states to revise their State Implementation Plans (SIPs) to include control measures to reduce emissions of SO₂ and/or NO_x. SO₂ is a precursor to $PM_{2.5}$ formation, and NO_x is a precursor to both ozone and $PM_{2.5}$ formation. Reducing upwind precursor emissions will assist the downwind $PM_{2.5}$ and 8-hour ozone nonattainment areas in achieving the NAAQS. Moreover, attainment would be achieved in a more equitable, cost-effective manner than if each nonattainment area attempted to achieve attainment by implementing local emissions reductions alone. The relevant regions for $PM_{2.5}$ and ozone significant contribution are depicted in Figure 2-1.

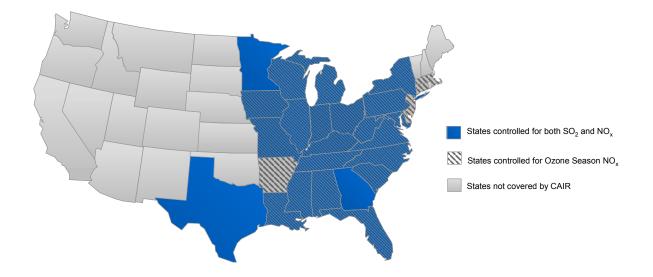


Figure 2-1. Final CAIR Region

The estimates presented in this report represent the benefits and costs for a final CAIR program that includes the final promulgated CAIR and the proposal to include SO_2 and annual NO_x controls for New Jersey and Delaware in CAIR. The modeling used to provide these estimates also assumes annual SO_2 and NO_x controls for Arkansas that are not a part of the complete CAIR program resulting in a slight overstatement of the reported benefits and costs.

2.3 Regulated Entities

This action does not directly regulate emissions sources. Instead, it requires states to revise their SIPs to include control measures to reduce emissions of NO_x and SO_2 . The emissions reduction requirements that would be assigned to the states are based on controls that are known to be highly cost-effective for EGUs. EPA modeled emission cap-and-trade programs phased in over time beginning with SO_2 and NO_x caps in 2010 and lowering these emission caps in 2015. The timing of emission caps was decided on the basis of when control actions would be needed to help the states in their NAAQS attainment efforts, feasibility of installing emission controls, and other factors. However, states would have the flexibility to choose the sources to control and how to control them. Although states have the flexibility to control pollution from sources other than EGUs, the analysis conducted assumes controls for EGUs only.

2.4 Baseline and Years of Analysis

The final rule on which this analysis is based sets forth the requirements for states to eliminate their significant contribution to downwind nonattainment of ozone and $PM_{2.5}$ NAAQS. To reduce this significant contribution, EPA requires that certain states reduce their emissions of SO₂ and NO_x. The Agency considered all promulgated CAA requirements and known state actions in the baseline used to develop the estimates of benefits and costs for this rule. EPA did not consider actions states may take to implement the ozone and $PM_{2.5}$ NAAQS standards in the baseline for this analysis. The years 2010 and 2015 are used in this analysis. The year of 2010 was chosen as one of the analysis years, because this year represents the date of Phase I of the rule and is in close proximity to the 2009 phase date for NO_x. The year 2015 represents the year in which Phase II of the rule is anticipated to be implemented. All estimates presented in this report represent annualized estimates of the benefits and costs of CAIR in 2010 and 2015 rather than the net present value of a stream of benefits and costs in these particular years of analysis.

2.5 Control Scenario

The analysis conducted assumes that a cap-and-trade program will be used to achieve the emission reduction requirements from the electric power industry. All fossil-fuel electric generating units (EGUs) over 25 megawatt (MW) capacity within the CAIR region are covered by the program. With the complete CAIR program (CAIR final plus the New Jersey and Delaware proposal), EPA would establish regional emission budgets (caps) for SO₂ and NO_x to address the transport problem. In this final rule, these requirements would effectively establish annual emission caps in 2010 for SO_2 and NO_x of 3.7 million tons and 1.5 million tons, respectively. These emission budgets (caps) would be lowered in 2015 to provide annual SO₂ and NO_x emission caps of 2.6 million tons and 1.3 million tons, respectively, in the control region. Banking of emissions is allowed in the program. These caps were derived by determining the amount of emissions of SO₂ and NO_x that EPA believes can be controlled from EGUs in a highly cost-effective manner. When fully implemented, this would result in nationwide SO_2 emissions of approximately 3.4 million tons. This is significantly lower than the 8.95 million tons of SO₂ emissions allowed under the current Title IV Acid Rain SO₂ Trading Program. For the final CAIR promulgated rule (exclusive of the New Jersey and Delaware proposal) emission caps are 3.6 million tons for SO₂ and 1.5 million tons for NO_x in 2010. These estimates become 2.5 million tons and 1.3 million tons in 2015.

2.6 Benefits of Emission Controls

The benefits of CAIR are discussed in Chapters 4 and 5 of this report. Annual monetized benefits of \$101 billion (3 percent discount rate) or \$86.3 billion (7 percent discount rate) are expected for CAIR in 2015. Despite the fact that the final CAIR program is comparable in most respects to the proposed rule, the benefits reported for the final rule exceed the estimates reported in the *Benefits of the Proposed Interstate Air Quality Rule* (OAR-2003-0053-0175, January 2004). There are several reasons for the increase in monetized benefit estimates for the final rule. These reasons include increased SO₂ emission reductions, geographical changes in the location of emission reductions with great reductions for the final rule.

2.7 Cost of Emission Controls

EPA analyzed the costs of CAIR using the Integrated Planning Model (IPM). EPA has used this model in the past to analyze the impacts of regulations on the power sector. EPA estimates the private industry costs of the rule to the power sector to be \$3.57 billion in 2015 (1999 dollars). In estimating the net benefits of the rule, EPA uses social costs of the rule that represent the costs to society of this rule. The social costs of the rule are estimated to be \$2.56 or \$3.07 billion in 2015 (3 percent and 7 percent discount rates, respectively). A description of the methodology used to model the costs and economic impacts to the power sector is discussed in Chapter 7 of this report.

2.8 Organization of the Regulatory Impact Analysis

This report presents EPA's analysis of the benefits, costs, and other economic effects of the final CAIR to fulfill the requirements of a Regulatory Impact Analysis (RIA). This RIA includes the following chapters:

- Chapter 3, Emissions and Air Quality Impacts, describes emission inventories and air quality modeling that are essential inputs into the benefits assessment.
- Chapter 4, Benefits Analysis and Results, describes the methodology and results of the benefits analysis.
- Chapter 5, Qualitative Assessment of Nonmonetized Benefits, describes benefits that are not monetized for this rulemaking.
- Chapter 6, Electric Power Sector Profile, describes the industry potentially affected by the rule.

- Chapter 7, Cost, Economic, and Energy Impacts of the Rule, describes the modeling conducted to estimate the cost, economic, and energy impacts to the affected sources.
- Chapter 8, Statutory and Executive Order Impact Analyses, describes the small business, unfunded mandates, paperwork reduction act, and other analyses conducted for the rule to meet statutory and Executive Order requirements.
- Chapter 9, Comparison of Benefits and Costs, shows a comparison of the social benefits to social costs of the rule.
- Appendix A, Benefits and Costs of the Clean Air Interstate Rule, Clean Air Visibility Rule, and the Clean Air Interstate Rule Plus the Clean Air Visibility Rule
- Appendix B, Supplemental Analyses Addressing Uncertainties in the Benefits Analyses
- Appendix C, Sensitivity Analyses of Key Parameters in the Benefits Analysis
- Appendix D, Sensitivity Analysis of Key Parameters in the Cost and Economic Impact Analysis and Listing of IPM Runs in Support of CAIR
- Appendix E, CAIR Industry Sector Impacts
- Appendix F, Additional Technical Information Supporting the Benefits Analysis
- Appendix G, Health-Based Cost-Effectiveness of Reductions in Ambient PM_{2.5} Associated With CAIR

SECTION 3

EMISSIONS AND AIR QUALITY IMPACTS

This chapter summarizes the emissions inventories and air quality modeling that serve as the inputs to the benefits analysis of this rule as detailed in Chapter 4. EPA uses sophisticated photochemical air quality models to estimate baseline and post-control ambient concentrations of ozone and PM and deposition of nitrogen and sulfur for each year. The estimated changes in ambient concentrations are then combined with monitoring data to estimate population-level exposures to changes in ambient concentrations for use in estimating health effects. Modeled changes in ambient data are also used to estimate changes in visibility and changes in other air quality statistics that are necessary to estimate welfare effects.

Section 3.1 of this chapter provides a summary of the baseline emissions inventories and the emissions reductions that were modeled for this rule. Section 3.2 provides a summary of the methods for and results of estimating air quality for the 2010 and 2015 base cases and control scenarios for the purposes of the benefits analysis. There are separate sections for PM, ozone, and visibility.

3.1 Emissions Inventories and Estimated Emissions Reductions

The technical support document for emissions inventories discusses the development of the 2001, 2010, and 2015 baseline emissions inventories for the benefits analysis of this final rule. The emission sources and the basis for current and future-year inventories are listed in Table 3-1. Tables 3-2 and 3-3 summarize the baseline emissions of NO_x and SO_2 and the change in the emissions from EGUs that were used in modeling air quality changes. For details on EPA's projected emissions for the EGU sector, see Chapter 7 of this RIA.

Sector	Emissions Source	2001 Base Year	Future-Year Base Case Projections
EGU	Power industry electric generating units (EGUs)	2001 data from Acid Rain Trading Program	Integrated Planning Model (IPM)
Non-EGU	Non-Utility Point, including point- source fugitive dust	2001 National Emission Inventory (NEI)	 Department of Energy (DOE) fuel use projections, (2) Regional Economic Model, Inc. (REMI) Policy Insight[®] model, (3) decreases to REMI results based on trade associations, Bureau of Labor Statistics (BLS) projections and Bureau of Economic Analysis (BEA) historical growth from 1987 to 2002, (4) control assumptions
Average Fire	Wildfire, prescribed burning	Same as future year	Average fires from 1996 through 2002 (based on state-total acres burned), with the same emissions rates and county distributions of emissions as in the 2001 NEI
Average Fire	Agricultural burning, open burning	2001 NEI	2001 NEI
Ag	Livestock NH ₃	2002 Preliminary NEI ^c	2010 and 2015 emissions estimated with the same approach as was used for the 2002 preliminary NEI ^e
Ag	Fertilizer NH ₃	2001 NEI	2001 NEI
Area	All other stationary area sources, including area- source fugitive dust	1999 NEI, version 3 grown to 2001	(1) DOE fuel use projections, (2) REMI Policy Insight model, (3) decreases to REMI results based on trade associations, BLS projections and BEA historical growth from 1987-2002
On-road	Highway vehicles	MOBILE6.2 model	Projected vehicle miles traveled same as IAQR proposal; emissions from MOBILE6.2 model
Nonroad	Locomotives, commercial marine vessels, and aircraft	2001 NEI; CMV adjusted to new national totals from OTAQ	Grown based on national totals from OTAQ, using state/county distribution of emissions from the 2001 NEI
Nonroad	All other nonroad vehicles	NONROAD2004 model	NONROAD 2004 model

Table 3-1. Emissions Sources and Basis for Current and Future-Year Inventories

^a This table documents only the sources of data for the U.S. inventory. The sources of data used for Canada and Mexico are explained in the technical support document (TSD) and were held constant from the base year to the future years.

^b All fugitive dust emissions were adjusted downward using county-specific transportable fractions needed as part of the current state of the art in air quality modeling.

^c ftp://ftp.epa.gov/EmisInventory/prelim2002nei/nonpoint/documentation/nh3inventorydraft_jan2004.pdf.

	Pollutant Emissions (tons)		
Source ^a	NO _X	SO ₂	
2001 Baseline			
EGUs	4,937,398	10,901,127	
Non-EGUs	2,942,618	2,958,692	
Average Fire	238,931	49,108	
Area	1,462,276	1,295,146	
On-road	8,064,067	271,026	
Nonroad	4,050,655	433,250	
Total, All Sources	21,695,945	15,908,349	
2010 Base Case			
EGUs	3,672,929	9,903,882	
Non-EGUs	2,931,360	3,189,864	
Average Fire	238,931	49,108	
Area	1,630,411	1,408,990	
Mobile	4,683,085	27,435	
Nonroad	3,282,338	219,029	
Total, All Sources	16,439,055	14,798,308	
2015 Base Case			
EGUs	3,708,658	9,079,214	
Non-EGUs	3,183,499	3,422,915	
Average Fire	238,931	49,108	
Area	1,702,154	1,480,348	
Mobile	3,152,562	30,824	
Nonroad	2,912,382	232,627	
Total, All Sources	14,898,186	14,295,035	

Table 3-2. Summary of Modeled Baseline Emissions for Lower 48 States

^a The "ag" sector does not have emissions of NO_x and SO_2 .

Table 3-3. Summary of Modeled Emissions Changes for the Clean Air Interstate Rule:2010 and 2015

	Pollutant	
Item	NO _X	SO ₂
2010 Emission Reductions ^a		
Absolute Tons	1,245,038	3,620,280
Percentage of Base EGU Emissions	33.9%	36.6%
Percentage of All Manmade Emissions	7.6%	24.5%
2015 Emission Reductions ^a		
Absolute Tons	1,535,821	3,967,777
Percentage of Base EGU Emissions	41.4%	43.7%
Percentage of All Manmade Emissions	10.3%	27.8%

^a Note that the emission changes only occur within the affected transport region; however, the percentage reductions reflect the change as a share of baseline emissions for the lower 48 states as presented in Table 3-2.

3.2 Air Quality Impacts

This section summarizes the methods for and results of estimating air quality for the 2010 and 2015 base cases and control scenarios for the purposes of the benefits analysis. EPA has focused on the health, welfare, and ecological effects that have been linked to air quality changes. These air quality changes include the following:

- 1. Ambient particulate matter (PM_{2.5})—as estimated using a national-scale applications of the <u>Community Multi-Scale Air Quality</u> (CMAQ) model;
- 2. Ambient ozone—as estimated using regional-scale applications of the <u>Comprehensive Air Quality Model with Extensions (CAMx); and</u>
- 3. Visibility degradation (i.e., regional haze), as developed using empirical estimates of light extinction coefficients and efficiencies in combination with CMAQ modeled reductions in pollutant concentrations.

The air quality estimates in this section are based on the emission changes summarized in the preceding section. These air quality results are in turn associated with human populations and ecosystems to estimate changes in health and welfare effects. In Section 3.2.1, we describe the estimation of PM air quality using CMAQ, and in Section 3.2.2, we cover the estimation of ozone air quality using CAMx. Lastly, in Section 3.2.3, we discuss the estimation of visibility degradation.

3.2.1 PM Air Quality Estimates

We use the emissions inputs summarized above with a national-scale application of the Community Multi-scale Air Quality (CMAQ) modeling system to estimate PM air quality in the contiguous United States. CMAQ is a three-dimensional grid-based Eulerian air quality model designed to estimate annual particulate concentrations and deposition over large spatial scales (e.g., over the contiguous United States). Consideration of the different processes that affect primary (directly emitted) and secondary (formed by atmospheric processes) PM at the regional scale in different locations is fundamental to understanding and assessing the effects of pollution control measures that affect PM, ozone, and deposition of pollutants to the surface.¹ Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, CMAQ is useful for evaluating the impacts of the rule on U.S. PM concentrations. Our analysis applies the modeling system to the entire United States for the six emissions scenarios: a 2001 base year, a 2010 baseline projection and a 2015 projection with controls, and a 2015 baseline projection and a 2015 projection with controls.

The CMAQ version 4.3 was employed for this CAIR modeling analysis (Byun and Schere, 2004). This version reflects updates in a number of areas to improve performance and address comments from the peer review, including (1) the formation of nitrates based on updated gaseous/heterogeneous chemistry and a current inorganic nitrate partitioning module, (2) a state-of-the-science Secondary Organic Aerosol (SOA) module that includes a more comprehensive gas-particle partioning algorithm from both anthropogenic and biogenic SOA, (3) an in-cloud sulfate chemistry that accounts for the nonlinear sensitivity of sulfate

¹Given the focus of this rule on secondarily formed particles (e.g., sulfates) it is important to employ a Eulerian model such as CMAQ. The formation and fate of secondarily formed pollutants typically involve emissions of precursor pollutants (e.g., SO₂) from a multitude of widely dispersed sources coupled with chemical and physical processes which are best addressed using an air quality model that employs a Eulerian grid model design.

formation to varying pH, and (4) the updated CB-IV gas-phase chemistry mechanism and aqueous chemistry mechanism that provide a comprehensive simulation of aerosol precursor oxidants.

CMAQ simulates every hour of every day of the year and, thus, requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include hourly emissions estimates and meteorological data in every grid cell, as well as a set of pollutant concentrations to initialize the model and to specify concentrations along the modeling domain boundaries. These initial and boundary concentrations were obtained from output of a global chemistry model. As discussed below, we use the model predictions in a relative sense by first determining the ratio of species predictions between the 2001 base year and each future-year scenario. The calculated relative change is then combined with the corresponding ambient species measurements to project concentrations for the future case scenarios. The annual mean PM air quality is used as input to the health and welfare concentration-response (C-R) functions of the benefits analysis. The following sections provide a more detailed discussion of each of the steps in this evaluation and a summary of the results.

3.2.1.1 Modeling Domain

As shown in Figure 3-1, the modeling domain encompasses the lower 48 states and extends from 126 degrees to 66 degrees west longitude and from 24 degrees north latitude to 52 degrees north latitude. The modeling domain is segmented into rectangular blocks referred to as grid cells. The model actually predicts pollutant concentrations for each of these grid cells. For this application the horizontal grid cells are roughly 36 km by 36 km. In addition, the modeling domain contains 14 vertical layers with the top of the modeling domain at about 16,200 meters, or 100 mb. Within the domain each vertical layer has 16,576 grid cells.

3.2.1.2 Simulation Periods

For use in this benefits analysis, the simulation periods modeled by CMAQ included separate full-year application for each of the five emissions scenarios (i.e., 2001 base year and the 2010 and 2015 base cases and control scenarios).

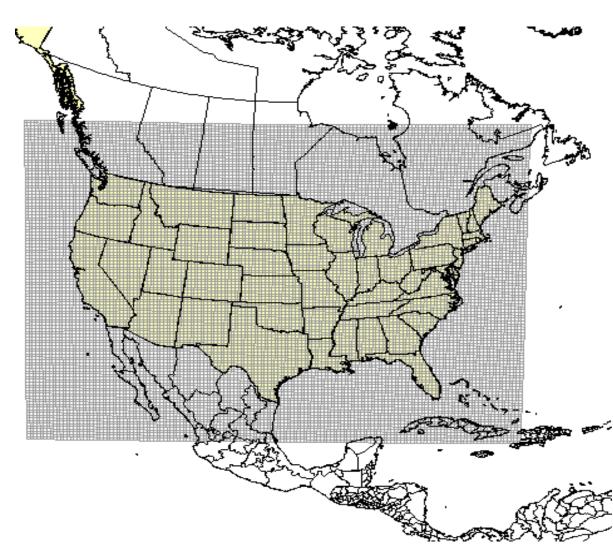


Figure 3-1. CMAQ Modeling Domain

3.2.1.3 Model Inputs

CMAQ requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, hourly emissions estimates and meteorological data and initial and boundary conditions. Separate emissions inventories were prepared for the 2001 base year and each of the future-year base cases and control scenarios. All other inputs were specified for the 2001 base year model application and remained unchanged for each future-year modeling scenario.

CMAQ requires detailed emissions inventories containing temporally allocated emissions for each grid cell in the modeling domain for each species being simulated. The previously described annual emission inventories were preprocessed into model-ready inputs through the emissions preprocessing system. Details of the preprocessing of emissions are provided in the Clean Air Interstate Rule Emissions Inventory Technical Support Document (Emissions Inventory TSD) (EPA, 2005). Meteorological inputs reflecting 2001 conditions across the contiguous United States were derived from version 5 of the Mesoscale Model (MM5). These inputs include horizontal wind components (i.e., speed and direction), temperature, moisture, vertical diffusion rates, and rainfall rates for each grid cell in each vertical layer.

The lateral boundary and initial species concentrations are provided by a threedimensional global atmospheric chemistry and transport model (GEOS-CHEM). The lateral boundary species concentrations varied with height and time (every 3 hours). Terrain elevations and land use information were obtained from the U.S. Geological Survey database at 10 km resolution and aggregated to the roughly 36 km horizontal resolution used for this CMAQ application.

3.2.1.4 CMAQ Model Evaluation

An operational model performance evaluation for PM_{2.5} and its related speciated components (e.g., sulfate, nitrate, elemental carbon, organic carbon) as well as deposition of ammonium, nitrate, and sulfate for 2001 was performed to estimate the ability of the CMAQ modeling system to replicate base-year concentrations. This evaluation principally comprises statistical assessments of model versus observed pairs that were paired in time and space on a daily or weekly basis, depending on the sampling period of measured data. The statistics are presented separately for the entire domain, the East, and the West (using the 100th meridian to divide the eastern and western United States). In addition, scatterplots of seasonal average and annual average predictions versus observations paired by site are included in the model performance evaluation. A spatial analysis was also performed for sulfate and nitrate to examine how well the modeling platform (year-specific meteorology, anthropogenic and biogenic emissions, and boundary conditions representative of 2001) predicts the spatial patterns and gradients evident from the observations. The details of these graphical analyses can be found in the Clean Air Interstate Rule Air Quality Modeling Technical Support Document (Air Quality Modeling TSD).

For $PM_{2.5}$ species, this evaluation includes comparisons of model predictions to the corresponding measurements from the <u>Clean Air Status and Trends Network (CASTNet)</u> and the <u>Speciation Trend Network (STN) in addition to measurements from the Interagency</u> <u>Monitoring of PRO</u>tected <u>Visual Environments (IMPROVE)</u>. The CASTNet dry deposition monitoring network contained a total of 79 sites in 2001, with a total number of 56 sites located in the East and 23 sites located in the West. Sulfate and total nitrate data were used in the evaluation. CASTNet data are collected and reported as weekly average data. The data are collected in filter packs that sample the ambient air continuously during the week. The sulfate data are of high quality because sulfate is a stable compound. However, the particulate nitrate concentration data collected by CASTNet are known to be problematic and subject to volatility because of the length of the sampling period. CASTNet also reports a total nitrate measurement is not affected by this sampling problem, it is considered a more reliable measurement. Therefore, we chose to use the total nitrate data and not to use the particulate nitrate data in this evaluation.

The EPA STN network began operation in 1999 to provide nationally consistent speciated $PM_{2.5}$ data for the assessment of trends at representative sites in urban areas. STN reports mass concentrations and $PM_{2.5}$ constituents, including sulfate, nitrate, ammonium, and elemental and organic carbon. Most STN sites collect data on a frequency of 1 in every 3 days, (some supplemental sites are collected 1 in every 6 days). For the 2001 analysis, CMAQ predictions were evaluated against 133 STN sites (105 sites in the East and 28 sites in the West).

The IMPROVE network is a cooperative visibility monitoring effort between EPA, federal land management agencies, and state air agencies. Data are collected at Class I areas across the United States mostly at national parks, national wilderness areas, and other protected pristine areas. Approximately 134 IMPROVE rural/remote sites had complete annual PM_{2.5} mass and/or PM_{2.5} species data for 2001. Eighty-six sites were in the West, and forty-eight sites were in the East. IMPROVE data are collected once in every 3 days.

The principal evaluation statistics used to evaluate CMAQ performance are the fractional bias and fractional error. Fractional bias is defined as:

$$FBLAS = \frac{2}{N} \sum_{i=1}^{N} \frac{(Pred_{x,t}^{i} - Obs_{x,t}^{i})}{(Pred_{x,t}^{i} + Obs_{x,t}^{i})} * 100$$

Fractional bias is a useful model performance indicator because it has the advantage of equally weighting positive and negative bias estimates. Fractional error is similar to fractional bias except the absolute value of the difference is used so that the error is always positive. Fractional error is defined as:

$$FERROR = \frac{2}{N} \sum_{i=1}^{N} \frac{|Pred_{x,t}^{i} - Obs_{x,t}^{i}|}{Pred_{x,t}^{i} + Obs_{x,t}^{i}} * 100$$

These metrics were calculated annually for all IMPROVE, CASTNet, STN, and <u>N</u>ational <u>A</u>tmospheric <u>DeP</u>osition (NADP) sites for the East and West individually.

Currently, there are no universally accepted performance criteria for judging the adequacy of PM_{2.5} model performance. However, performance can be judged by comparison to model performance results found by other groups in the air quality modeling community. In this respect, we have compared our CMAQ 2001 model performance results to the range of performance found in other recent regional PM_{2.5} model applications by other groups. These modeling studies represent a broad range of modeling analyses that cover various models, model configurations, domains, years and/or episodes, chemical mechanisms, and aerosol modules. The fractional bias and fractional error statistics were calculated using the predicted-observed pairs for the full year of 2001 and for each season, separately. The statistics for the full year are provided in Table 3-4. Overall, the performance is within the range or close to that found by other groups in recent applications. It should be noted that nitrate and sulfate are the two species most relevant for CAIR. Model performance statistics for these two species during the seasons when the concentrations are highest in the East (i.e., summer for sulfate and winter for nitrate) are provided in Table 3-5. The general range of model performance for summer sulfate and winter nitrate compares favorably to fractional bias and fractional error statistics from the better performing model applications found by others in the modeling community, as follows:

CAIR CMAQ 2001 A	nnual		Fractional Bias (%)	Fractional Error (%)
	STN	East	-12	44
PM _{2.5}		West	-51	64
Total Mass	IMPROVE	East	8	43
		West	14	57
	STN	East	8	45
		West	-32	52
0-16-1-	IMPROVE	East	7	40
Sulfate		West	-2	49
	CASTNet	East	-2	24
		West	-35	50
	STN	East	-12	86
		West	-92	115
Nitrate	IMPROVE	East	-32	107
		West	-44	115
Total Nitrate	CASTNet	East	16	81
$(NO_3 + HNO_3)$		West	-60	105
	STN	East	32	63
El mantel Centera		West	-20	67
Elemental Carbon	IMPROVE	East	-23	51
		West	-13	66
	STN	East	-3	75
Onequie Canherr		West	-31	66
Organic Carbon	IMPROVE	East	-10	52
		West	51	76

Table 3-4. Model Performance Statistics for CAIR CMAQ 2001

- summer sulfate is in the range of -10 percent to +30 percent for fractional bias and 35 percent to 50 percent for fractional error and
- winter nitrate is in the range of +50 percent to +70 percent for fractional bias and 85 percent to 105 percent for fractional error.

Thus, CMAQ is considered appropriate for use in projecting changes in future year $PM_{2.5}$ concentrations and the resultant health/economic benefits due to the emissions reductions.

Eastaun	United States	СМА	Q 2001
Lastern	United States	Fractional Bias (%)	Fractional Error (%)
0.10.	STN	14	44
Sulfate (Summer)	IMPROVE	10	42
(Summer)	CASTNet	3	22
Nitrate	STN	15	73
(Winter)	IMPROVE	21	92

 Table 3-5. Selected Performance Evaluation Statistics from the CMAQ 2001

 Simulation

3.2.1.5 Converting CMAQ Outputs to Benefits Inputs

CMAQ generates predictions of hourly PM species concentrations for every grid. The species include a primary coarse fraction (corresponding to PM in the 2.5 to 10 micron size range), a primary fine fraction (corresponding to PM less than 2.5 microns in diameter), and several secondary particles (e.g., sulfates, nitrates, and organics). PM_{2.5} is calculated as the sum of the primary fine fraction and all of the secondarily formed particles. Future-year estimates of PM_{2.5} were calculated using relative reduction factors (RRFs) applied to 2002 ambient PM_{2.5} and PM_{2.5} species concentrations. A gridded field of PM_{2.5} concentrations was created by interpolating Federal Reference Monitor ambient data and IMPROVE ambient data. Gridded fields of PM_{2.5} species concentrations were created by interpolating EPA speciation network (ESPN) ambient data and IMPROVE data. The ambient data were interpolated to the CMAQ 36 km grid.

The procedures for determining the RRFs are similar to those in EPA's draft guidance for modeling the $PM_{2.5}$ standard (EPA, 2000). This guidance has undergone extensive peer review and is anticipated to be finalized this year. The guidance recommends that model predictions be used in a relative sense to estimate changes expected to occur in each major $PM_{2.5}$ species. The procedure for calculating future-year $PM_{2.5}$ design values is called the "Speciated Modeled Attainment Test (SMAT)." EPA used this procedure to estimate the ambient impacts of the CAIR NPR emissions controls. The SMAT procedures for the No Further Remediation (NFR) have been revised. Full documentation of the revised SMAT methodology is contained in the Air Quality Modeling TSD.

The revised SMAT uses an FRM mass construction methodology that results in reduced nitrates (relative to the amount measured by routine speciation networks, such as ESPN), higher mass associated with sulfates (reflecting water included in FRM measurements), and a measure of organic carbonaceous mass that is derived from the difference between measured $PM_{2.5}$ and its noncarbon components. This characterization of $PM_{2.5}$ mass also reflects crustal material and other minor constituents. The resulting characterization provides a complete mass balance. It does not have any unknown mass that is sometimes presented as the difference between measured $PM_{2.5}$ mass and the characterized chemical components derived from routine speciation measurements. The revised SMAT methodology uses the following $PM_{2.5}$ species components: sulfates, nitrates, ammonium, organic carbon mass, elemental carbon, crustal, water, and blank mass (a fixed value of 0.5 ug/m³). In each grid cell, the $PM_{2.5}$ component species mass adds up to interpolated $PM_{2.5}$ mass.

For the purposes of projecting future $PM_{2.5}$ concentrations for input to the benefits calculations, we applied the SMAT procedure using the base-year 2001 modeling scenario and each of the future-year scenarios. In our application of SMAT we used temporally scaled speciated $PM_{2.5}$ monitor data from 2002 as the set of base-year measured concentrations. Temporal scaling is based on ratios of model-predicted future case $PM_{2.5}$ species concentrations to the corresponding model-predicted 2001 concentrations. Output files from this process include both quarterly and annual mean $PM_{2.5}$ mass concentrations, which are then manipulated within SAS to produce a BenMAP input file containing 364 daily values (created by replicating the quarterly mean values for each day of the appropriate season).

The SMAT procedures documented in the Air Quality Modeling TSD are applicable for projecting future nonattainment counties and downwind receptor areas for the transport analysis. Those procedures are the same as those performed for the PM benefits analysis with the following exceptions:

1) The benefits analysis uses interpolated $PM_{2.5}$ data that cover all of the grid cells in the modeling domain (covering the entire country), whereas the nonattainment analysis is performed at each ambient monitoring site in the East using measured $PM_{2.5}$ data (only the species data are interpolated).

The benefits analysis is anchored by the interpolated PM_{2.5} data from the single year of 2002, whereas the nonattainment analysis uses a 5-year weighted average (1999–2003) of PM_{2.5} design values at each monitoring site.

3.2.1.6 PM Air Quality Results

Table 3-6 summarizes the projected $PM_{2.5}$ concentrations for the 2010 and 2015 base cases and changes associated with the rule. The table includes the annual mean concentration averaged across all model grid cells in the East and West,² separately, along with the average change between base and control concentrations. We also provide the population-weighted average that better reflects the baseline levels and predicted changes for more populated areas of the nation. This measure, therefore, better reflects the potential benefits of these predicted changes through exposure changes to these populations. As shown, the average annual mean concentrations of $PM_{2.5}$ across populated eastern U.S. grid cells declines by roughly 7.1 percent (or 0.73 µg/m³) and 8.7 percent (or 0.89 µg/m³) in 2010 and 2015, respectively. The population-weighted average mean concentration declined by 8.1 percent (or 0.96 µg/m³) in 2010 and 9.8 percent (or 1.15 µg/m³) in 2015, and this change is larger in absolute terms than the spatial average for both years. This indicates the rule generates greater absolute air quality improvements in more populated urban areas.

Table 3-7 provides information on the populations in 2010 and 2015 that will experience improved PM air quality. Significant populations live in areas with meaningful reductions in annual mean $PM_{2.5}$ concentrations resulting from the rule. As shown, in 2015, almost 63 percent of the U.S. population located in the eastern 37-state modeling domain is predicted to experience reductions of greater than 0.5 µg/m³. This is an increase from the 54 percent of the U.S. population that is expected to experience such reductions in 2010. Furthermore, over 40 percent of this population will benefit from reductions in annual mean $PM_{2.5}$ concentrations of greater than 1 µg/m³, and almost 23 percent will live in areas with reductions of greater than 1.5 µg/m³.

²For the purpose of this analysis "East" is defined as the U.S. portion of the modeling domain east of 100 degrees longitude, and similarly "West" is defined as the U.S. portion of the domain west of 100 degrees longitude.

			2010			2015	
Region	PM _{2.5} (µg/m ³)	Base Case	Change ^a	Percent Change	Base Case	Change ^a	Percent Change
	Average ^b Annual Mean	10.36	-0.73	-7.1	10.28	-0.89	-8.7
East	Population-Weighted Average Annual Mean	11.91	-0.96	-8.1	11.79	-1.15	-9.8
	Average Annual Mean	6.04	-0.02	-0.3	6.07	-0.03	-0.5
West	Population-Weighted Average Annual Mean ^c	12.38	-0.01	-0.1	12.50	-0.01	-0.1

Table 3-6. Summary of Base Case PM Air Quality and Changes Due to Clean AirInterstate Rule: 2010 and 2015

^a The change is defined as the control case value minus the base case value.

^b Calculated as the average across all grid cells in the U.S. portion of the region.

^c Calculated by summing the product of the population and the projected annual mean PM concentration for each grid cell then dividing this sum by the total population.

3.2.2 Ozone Air Quality Estimates

We use the emissions inputs summarized earlier in this chapter with a regional-scale application of CAMx to estimate ozone air quality in the East. CAMx is a Eulerian threedimensional photochemical grid air quality model designed to calculate the concentrations of both inert and chemically reactive pollutants by simulating the physical and chemical processes in the atmosphere that affect ozone formation. Version 3.10 of the CAMx model was employed for these analyses. Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, CAMx is useful for evaluating the impacts of the final rule on ozone concentrations.

Our analysis applies the modeling system separately to the Eastern United States for five emissions scenarios: a 2001 baseline, a 2010 baseline projection and a 2010 projection with controls, and a 2015 baseline projection and a 2015 projection with controls. Further discussion of this modeling, including an evaluation of model performance relative to observed ozone, is provided in the Air Quality Modeling TSD. As discussed in Chapter 4,

	2010 Po	pulation ^b	2015 Population		
Change in Annual Mean PM _{2.5} Concentrations (μg/m³) ^a	Number (millions)	Percent (%)	Number (millions)	Percent (%)	
$\Delta \ PM_{2.5} \ Conc \leq 0.25$	81.7	26.8%	80.8	26.5%	
$0.25 > \Delta PM_{2.5} Conc \le 0.5$	58.4	19.2%	31.9	10.5%	
$0.5 > \Delta PM_{2.5} Conc \le 0.75$	36.2	11.9%	54.8	18.0%	
$0.75 > \Delta PM_{2.5} Conc \le 1.0$	24.0	7.9%	26.1	8.6%	
$1.0 > \Delta PM_{2.5} Conc \le 1.25$	41.1	13.5%	16.3	5.4%	
$1.25 > \Delta PM_{2.5} Conc \le 1.5$	21.8	7.2%	37.5	12.3%	
$1.5 > \Delta PM_{2.5} Conc \le 1.75$	14.5	4.8%	26.4	8.7%	
$1.75 > \Delta PM_{2.5} Conc \le 2.0$	8.9	2.9%	16.8	5.5%	
$\Delta PM_{2.5} Conc > 2.0$	17.7	5.8%	26.2	8.6%	

Table 3-7. Distribution of PM2.5 Air Quality Improvements Over Population Due toClean Air Interstate Rule: 2010 and 2015

^a The change is defined as the control case value minus the base case value.

^b Population counts and percentages are for the fraction of the continental U.S. population located in the modeling domain considered in modeling health benefits for the rule.

we use the relative predictions from the model by combining the 2001 base-year and each future-year scenario with current ambient air quality observations to determine the expected change in 2010 or 2015 ozone concentrations due to the rule. These results are used solely in the benefits analysis.

The CAMx modeling system requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, day-specific emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. As applied to the Eastern United States, the model segments the area into square blocks called grids (roughly equal in size to counties), each of which has several layers of air conditions that are considered in the analysis. Using these data, the CAMx model generates predictions of hourly ozone concentrations for every grid. We used the results of this process to develop 2010 and 2015 ozone profiles at monitor sites by normalizing the CAMx predictions to the observed ozone concentrations at each monitor site. For areas (grids) without ozone monitoring data, we interpolated ozone values using data from monitors surrounding the area. After completing this process, we calculated daily

and seasonal ozone metrics to be used as inputs to the health and welfare C-R functions of the benefits analysis. The following sections provide a more detailed discussion of each of the steps in this evaluation and a summary of the results.

3.2.2.1 Modeling Domain

The modeling domain representing the Eastern United States is the same as that used previously for Ozone Transport Assessment Group and for the On-highway Tier-2 rulemaking. As shown in Figure 3-2, this domain encompasses most of the Eastern United States from the East coast to mid-Texas and consists of two grids with differing resolutions. The modeling domain extends from 99 degrees to 67 degrees west longitude and from 26 degrees to 47 degrees north latitude. The inner portion of the modeling domain shown in Figure 3-2 uses a relatively fine grid of approximately 12 km consisting of nine vertical layers. The outer area has less horizontal resolution. The grid cell size in the outer grid is approximately 36 km with the same nine vertical layers. The vertical height of the modeling domain is 4,000 meters above ground level for both areas.

3.2.2.2 Simulation Periods

For use in this benefits analysis, the simulation periods modeled by CAMx included several multiday periods when ambient measurements recorded high ozone concentrations. A simulation period, or episode, consists of meteorological data characterized over a block of days that are used as inputs to the air quality model. We modeled three periods during the summer of 1995: June 12–24, July 5–15, and August 7–21. Collectively, these periods contain episodes of high ozone in various portions of the East. The six emissions scenarios (1995 base year, 2001 base year, 2010 base and control, 2015 base and control) were simulated for all three episodes. The periods modeled include three "ramp-up" days to initialize the model, but the results for these days are not used in this analysis.

3.2.2.3 Nonemissions Modeling Inputs

The meteorological data required for input into CAMx (e.g., wind, temperature, vertical mixing) were developed by separate meteorological models. The gridded meteorological data for the three historical 1995 episodes were developed using the Regional Atmospheric Modeling System (RAMS), version 3b. This model provided needed data at every grid cell on an hourly basis. These meteorological modeling results were evaluated against observed weather conditions before being input into CAMx, and it was concluded that the model fields were adequate representations of the historical meteorology. A more

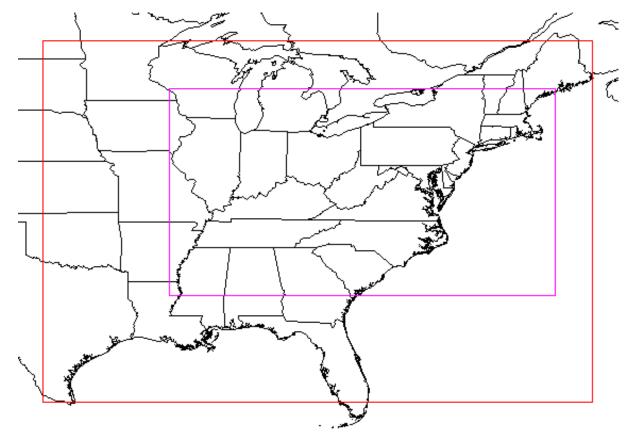


Figure 3-2. CAMx Eastern U.S. Modeling Domain

Note: The inner area represents fine grid modeling at 12 km resolution. The outer area represents the coarse grid modeling at 36 km resolution.

detailed description of the settings and assorted input files employed in these applications is provided in the Air Quality Modeling TSD.

The modeling assumed background pollutant levels at the top and along the periphery of the domain. Initial conditions were also assumed to be relatively clean. Given the rampup days and the expansive domains, it is expected that these assumptions will not affect the modeling results. The development of model inputs is discussed in greater detail in the Air Quality Modeling TSD, which is available in the docket for this rule.

3.2.2.4 Model Performance for Photochemical Ozone

A performance evaluation of CAMx for the three 1995 episodes was conducted prior to CAIR, in support of the Nonroad Diesel Engine Rule. A summary of model performance

from the study is provided here. In this analysis, a series of performance statistics was calculated for the Eastern U.S. domain as well as the four quadrants of this domain and multiple subregions. The model performance evaluation consisted solely of comparisons against ambient surface ozone data.

Three primary statistical metrics were used to assess the overall accuracy of the baseyear modeling simulations:

- Mean normalized bias is defined as the average difference between the hourly model predictions and observations (paired in space and time) at each monitoring location, normalized by the magnitude of the observations.
- Mean normalized gross error is defined as the average absolute difference between the hourly model predictions and observations (paired in space and time) at each monitoring location, normalized by the magnitude of the observations.
- Average accuracy of the peak is defined as the average difference between peak daily model predictions and observations at each monitoring location, normalized by the magnitude of the observations.

In general, the model tends to slightly underestimate observed ozone. When all hourly observed ozone values greater than a 60 ppb threshold are compared to their model counterparts for the 30 episode modeling days in the eastern domain, the mean normalized bias is -1.1 percent and the mean normalized gross error is 20.5 percent. As shown in Table 3-8, the model generally underestimates observed ozone values for the June and July episodes but predicts higher than observed amounts for the August episode.

Episode	Average Accuracy of the Peak	Mean Normalized Bias	Mean Normalized Gross Error
June 1995	-7.3	-8.8	19.6
July 1995	-3.3	-5.0	19.1
August 1995	9.6	8.6	23.3

Table 3-8. Model Performance Statistics for Hourly Ozone in the Eastern U.S. CAMx
Ozone Simulations: 1995 Base Case

At present, there are no guidance criteria by which one can determine if a regional ozone modeling exercise is exhibiting adequate model performance. These base-case simulations were determined to be acceptable based on comparisons to previously completed model rulemaking analyses (e.g., OTAG, Tier-2, and Heavy-Duty Engine). The modeling completed for this rule exhibits less bias and error than any past regional ozone modeling application done by EPA. Thus, the model is considered appropriate for use in projecting changes in future-year ozone concentrations and the resultant health/economic benefits due to the emissions reductions.

In addition, the CAMx modeling results were also evaluated at a "local" level to ensure that areas determined to need the emissions reductions based on projected exceedances of the ozone standard were not unduly influenced by local overestimation of ozone in the model base year. As detailed in the Air Quality Modeling TSD, performance statistics were computed for each of 51 local subregions within the modeling domain. These performance statistics were compared to the recommended performance ranges for urban attainment modeling (EPA, 1999). The results indicate that model performance for the June episode was within the recommended ranges for 69 percent of the local areas examined. For the July and August episodes, the percentage of local areas with performance within the recommended ranges was 80 percent and 61 percent, respectively.

3.2.2.5 Converting CAMx Outputs to Full-Season Profiles for Benefits Analysis

This study extracted hourly, surface-layer ozone concentrations for each grid cell from the standard CAMx output file containing hourly average ozone values. These model predictions are used in conjunction with the observed concentrations obtained from the Aerometric Information Retrieval System (AIRS) to generate ozone concentrations for the entire ozone season.^{3,4} The predicted changes in ozone concentrations from the future-year base case to future-year control scenario serve as inputs to the health and welfare C-R functions of the benefits analysis (i.e., the Environmental <u>Ben</u>efits <u>Mapping and Analysis</u> <u>Program [BenMAP]</u>).

³The ozone season for this analysis is defined as the 5-month period from May to September; however, to estimate certain crop yield benefits, the modeling results were extended to include months outside the 5-month ozone season.

⁴Based on AIRS, there were 961 ozone monitors with sufficient data (i.e., 50 percent or more days reporting at least nine hourly observations per day [8 am to 8 pm] during the ozone season).

To estimate ozone-related health and welfare effects for the contiguous United States, full-season ozone data are required for every BenMAP grid-cell. Given available ozone monitoring data, we generated full-season ozone profiles for each location in the contiguous 48 states in two steps: (1) we combined monitored observations and modeled ozone predictions to interpolate hourly ozone concentrations to a grid of 8 km by 8 km population grid cells, and (2) we converted these full-season hourly ozone profiles to an ozone measure of interest, such as the daily average.^{5,6}

3.2.2.6 Ozone Air Quality Results

This section provides a summary of the predicted ambient ozone concentrations from the CAMx model for the 2010 and 2015 base cases and changes associated with the rule. Table 3-9 provides those ozone metrics for grid cells in the Eastern United States that enter the C-R functions for health benefits endpoints. The population-weighted average reflects the baseline levels and predicted changes for more populated areas of the nation. This measure, therefore, will better reflect the potential benefits of these predicted changes through exposure changes to these populations.

3.2.3 Visibility Degradation Estimates

Visibility degradation is often directly proportional to decreases in light transmittal in the atmosphere. Scattering and absorption by both gases and particles decrease light transmittance. To quantify changes in visibility, our analysis computes a light-extinction coefficient, based on the work of Sisler (1996), which shows the total fraction of light that is decreased per unit distance. This coefficient accounts for the scattering and absorption of light by both particles and gases and accounts for the higher extinction efficiency of fine particles compared to coarse particles. Fine particles with significant light-extinction efficiencies include sulfates, nitrates, organic carbon, elemental carbon (soot), and soil (Sisler, 1996).

⁵The 8-km grid squares contain the population data used in the health benefits analysis model, BenMAP. See Chapter 4 for a discussion of this model.

⁶This approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (EVNA) spatial interpolation.

 Table 3-9.
 Summary of CAMx Derived Population-Weighted Ozone Air Quality

 Metrics for Health Benefits Endpoints Due to Clean Air Interstate Rule:
 Eastern U.S.

	2010			2015		
Statistic ^a	Base Case	Change ^b	Percent Change ^b	Base Case	Change ^b	Percent Change ^b
Population-Weighted Average (ppb) ^d						
Daily 1-Hour Maximum Concentration	51.82	-0.50	-1.0%	50.77	-1.36	-2.7%
Daily 8-Hour Average Concentration	42.59	-0.39	-1.0%	41.84	-1.05	-2.5%
Daily 12-Hour Average Concentration	40.09	-0.37	-1.0%	39.41	-0.97	-2.5%
Daily 24-Hour Average Concentration	30.15	-0.27	-1.0%	29.73	-0.67	-2.3%

^a These ozone metrics are calculated at the CAMX grid-cell level for use in health effects estimates based on the results of spatial and temporal Voronoi Neighbor Averaging. Except for the daily 24-hour average, these ozone metrics are calculated over relevant time periods during the daylight hours of the "ozone season" (i.e., May through September). For the 8-hour average, the relevant time period is 9 am to 5 pm, and for the 12-hour average it is 8 am to 8 pm.

^b The change is defined as the control-case value minus the base-case value. The percentage change is the "Change" divided by the "Base Case" and then multiplied by 100 to convert the value to a percentage.

^d Calculated by summing the product of the projected CAMx grid-cell population and the estimated CAMx grid cell seasonal ozone concentration and then dividing by the total population.

Based on the light-extinction coefficient, we also calculated a unitless visibility index, called a "deciview," which is used in the valuation of visibility. The deciview metric provides a scale for perceived visual changes over the entire range of conditions, from clear to hazy. Under many scenic conditions, the average person can generally perceive a change of one deciview. The higher the deciview value, the worse the visibility. Thus, an improvement in visibility is a decrease in deciview value.

Table 3-10 provides visibility improvements expected to occur in specific parks in the CAIR region. As shown, major parks in the Eastern United States, including the Great Smokey Mountains and Shenandoah, are expected to see significant improvements in visibility. By 2015, on the 20 percent of worst visibility days, the Great Smokey Mountains National Park is expected to see improvements of over 2.5 deciviews (9 percent), and Shenandoah National Park is expected to see improvements of over 3.3 deciviews (12 percent). Under average light conditions, these represent improvements in visual range by close to 7 miles in the Great Smokies and over 10 miles in Shenandoah.

		201	0		2015			
Federal Class I Area	Change in Average of 20% Worst Davs	Percent Change in Average of 20% Worst Davs	Change in Annual Average	Percent Change in Annual Average	Change in Average of 20% Worst Davs	Percent Change in Average of 20% Worst Days	Change in Annual Average	Percent Change in Annual Average
Acadia, ME	0.88	3.98	0.36	2.59	1.00	4.54	0.42	2.94
Boundary Waters Canoe Area, MN	0.26	1.36	0.12	0.95	0.29	1.52	0.12	0.94
Brigantine, NJ	1.88	6.90	0.93	4.59	2.07	7.54	1.04	4.97
Caney Creek, AR	1.08	4.30	0.42	2.17	1.32	5.24	0.54	2.80
*Chassahowitzka, FL	0.90	3.76	0.47	2.45	1.66	6.92	1.03	5.38
*Dolly Sods, WV	2.39	9.03	1.44	7.41	2.75	10.54	1.68	8.71
*Everglades, FL	0.42	2.13	0.17	1.16	0.49	2.43	0.21	1.35
Great Gulf, NH	1.32	5.87	0.44	3.10	1.56	6.95	0.50	3.53
*Great Smoky Mountains, TN	1.85	6.40	0.97	4.65	2.61	9.12	1.36	6.58
Isle Royale, MI	0.31	1.42	0.17	1.29	0.38	1.76	0.18	1.41
*James River Face, VA	2.09	7.55	1.21	5.93	2.45	8.96	1.41	6.94
*Joyce Kilmer—Slickrock, TN	1.85	6.40	0.97	4.65	2.61	9.12	1.36	6.58
*Linville Gorge, NC	1.71	6.22	1.00	5.18	2.14	7.92	1.28	6.65
Lye Brook, VT	1.76	7.20	0.61	4.31	2.10	8.64	0.72	5.07
*Mammoth Cave, KY	1.68	5.61	0.94	4.20	2.45	8.31	1.32	5.96
Mingo, MO	0.82	2.97	0.50	2.41	0.95	3.46	0.58	2.85
Moosehorn, ME	0.82	3.83	0.30	2.10	0.92	4.30	0.33	2.30
*Okefenokee, GA	0.99	3.87	0.63	3.21	1.44	5.64	0.93	4.70

Table 3-10. Summary of Deciview Visibility Impacts at Class I Areas in the CAIR Region^{a,b}

		201	0		2015			
Federal Class I Area	Change in Average of 20% Worst Days	Percent Change in Average of 20% Worst Days	Change in Annual Average	Percent Change in Annual Average	Change in Average of 20% Worst Days	Percent Change in Average of 20% Worst Days	Change in Annual Average	Percent Change in Annual Average
*Otter Creek, WV	2.47	9.22	1.45	7.43	2.94	11.14	1.72	8.90
Presidential Range - Dry, NH	1.50	6.70	0.52	3.75	1.76	7.93	0.60	4.28
*Cape Romain, SC	1.01	4.17	0.77	4.07	1.44	5.98	1.06	5.58
Roosevelt Campobello, ME	0.80	3.76	0.29	2.03	0.94	4.41	0.33	2.30
Seney, MI	0.65	2.64	0.23	1.67	0.78	3.16	0.25	1.84
*Shenandoah, VA	2.81	10.23	1.69	8.54	3.31	12.27	2.01	10.24
*Sipsey, AL	1.45	5.27	0.81	3.83	2.06	7.55	1.08	5.11
*Swanquarter, NC	1.45	6.04	0.80	4.39	1.86	7.85	1.00	5.54
Upper Buffalo, AR	0.67	2.74	0.31	1.72	0.80	3.27	0.39	2.17
Voyageurs, MN	0.12	0.68	0.09	0.74	0.12	0.72	0.08	0.65
*Wolf Island, GA	0.84	3.28	0.60	3.06	1.12	4.37	0.79	3.99

Table 3-10. Summary of Deciview Visibility Impacts at Class I Areas in the CAIR Region (continued)

^a The change is defined as the base case value minus the control case value.

^b The percent change is the "Change" divided by the "Base Case" and then multiplied by 100 to convert the value to a percentage.

* Visibility Benefits were monitized for this park.

3.3 References

- Byun, D., and K.L. Schere. March 2004. "Review of the Governing Equations, Computational Algorithms, and Other Components of the Models-3 Community Multiscale Air Quality (CMAQ) Modeling System." Submitted to the *Journal of Applied Mechanics Reviews*.
- Sisler, J.F. July 1996. Spatial and Seasonal Patterns and Long Term Variability of the Composition of the Haze in the United States: An Analysis of Data from the IMPROVE Network. Fort Collins, CO: Cooperative Institute for Research in the Atmosphere, Colorado State University.
- U.S. Environmental Protection Agency (EPA). 1999. Draft Guidance on the Use of Models and Other Analyses in Attainment Demonstrations for the 8-Hour Ozone NAAQS, Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. Environmental Protection Agency (EPA). 2000. Draft Guidance for Demonstrating Attainment of the Air Quality Goals for PM_{2.5} and Regional Haze; Draft 1.1, Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. Environmental Protection Agency (EPA). 2005. Clean Air Interstate Rule Air Quality Modeling Technical Support. Office of Air Quality Planning and Standards. Research Triangle Park, N.C.
- U.S. Environmental Protection Agency (EPA). 2005. Clean Air Interstate Rule Emission Inventory Technical Support Document. Office of Air Quality Planning and Standards. Research Triangle Park, NC.

CHAPTER 4

BENEFITS ANALYSIS AND RESULTS

This chapter reports EPA's analysis of a subset of the public health and welfare impacts and associated monetized benefits to society of CAIR. EPA is required by Executive Order (E.O.) 12866 to estimate the benefits and costs of major new pollution control regulations. Accordingly, the analysis presented here attempts to answer three questions: (1) what are the physical health and welfare effects of changes in ambient air quality resulting from reductions in precursors to particulate matter (PM) including NO_x and SO₂ emissions? (2) what is the monetary value of the changes in these effects attributable to the final rule? and (3) how do the monetized benefits compare to the costs? It constitutes one part of EPA's thorough examination of the relative merits of this regulation.

The analysis presented in this chapter uses a methodology generally consistent with benefits analyses performed for the recent analysis of Nonroad Diesel Engines Tier 4 Standards and the proposed Clear Skies Act of 2003 (EPA, 2003c). The benefits analysis relies on three major modeling components:

- 1) Calculation of the impact of CAIR on EGUs assuming a cap-and-trade program based on the national inventory of precursors to PM, specifically NO_x and SO₂.
- Air quality modeling for 2010 and 2015 to determine changes in ambient concentrations of ozone and PM, reflecting baseline and postcontrol emissions inventories.
- A benefits analysis to determine the changes in human health and welfare, both in terms of physical effects and monetary value, that result from the projected changes in ambient concentrations of various pollutants for the modeled standards.

A wide range of human health and welfare effects are linked to the emissions of NO_x and SO_2 from EGUs and the resulting impact on ambient concentrations of ozone and PM. Potential human health effects associated with $PM_{2.5}$ range from premature mortality to morbidity effects linked to long-term (chronic) and shorter-term (acute) exposures (e.g., respiratory and cardiovascular symptoms resulting in hospital admissions, asthma

exacerbations, and acute and chronic bronchitis [CB]). Exposure to ozone has also been linked to a variety of respiratory effects including hospital admissions and illnesses resulting in school absences. Some studies, including a recent multi-city analysis of 95 major U.S. urban areas (Bell et al., 2004), have linked short term ozone exposures with premature mortality.¹ Welfare effects potentially linked to PM include materials damage and visibility impacts, while ozone can adversely affect the agricultural and forestry sectors by decreasing yields of crops and forests. Although methods exist for quantifying the benefits associated with many of these human health and welfare categories, not all can be evaluated at this time because of limitations in methods and/or data. Table 4-1 summarizes the annual monetized health and welfare benefits associated with CAIR for 2 years, 2010 and 2015. Table 4-2 lists the full complement of human health and welfare effects associated with PM and ozone and identifies those effects that are quantified for the primary estimate and those that remain unquantified because of current limitations in methods or available data.

Figure 4-1 illustrates the major steps in the benefits analysis. Given baseline and post-control emissions inventories for the emission species expected to affect ambient air quality, we use sophisticated photochemical air quality models to estimate baseline and post-control ambient concentrations of ozone and PM and deposition of nitrogen and sulfur for each year. The estimated changes in ambient concentrations are then combined with

	Total Benefits ^{a, b} (b	illions 1999\$)
	2010	2015
Using a 3% discount rate	\$73.3 + B	\$101 + B

Table 4-1. Estimated Monetized Benefits of the Final CAIR

Using a 7% discount rate

^a For notational purposes, unquantified benefits are indicated with a "B" to represent the sum of additional monetary benefits and disbenefits. A detailed listing of unquantified health and welfare effects is provided in Table 4-2.

62.6 + B

86.3 + B

^b Results reflect the use of two different discount rates: 3 and 7 percent, which are recommended by EPA's *Guidelines for Preparing Economic Analyses* (EPA, 2000b) and OMB Circular A-4 (OMB, 2003). Results are rounded to three significant digits for ease of presentation and computation.

¹Short-term exposure to ambient ozone has also been linked to premature death. EPA is currently evaluating the epidemiological literature examining the relationship between ozone and premature mortality, sponsoring three independent meta-analyses of the literature. EPA will consider including ozone mortality in primary benefits analyses once a peer-reviewed methodology is available.

Pollutant/Effect	Quantified and Monetized in Base Estimates ^a	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects - Changes in:
Ozone/Health ^b	Hospital admissions: respiratory Emergency room visits for asthma Minor restricted-activity days School loss days	Premature mortality: short term exposures ^e Asthma attacks Cardiovascular emergency room visits Acute respiratory symptoms	Chronic respiratory damage Premature aging of the lungs Nonasthma respiratory emergency room visits Increased exposure to UVb
Ozone/Welfare	Decreased outdoor worker productivity		 Yields for: Commercial forests Fruits and vegetables, and Other commercial and noncommercial crops Damage to urban ornamental plants Recreational demand from damaged forest aesthetics Ecosystem functions Increased exposure to UVb

Table 4-2. Human Health and Welfare Effects of Pollutants Affected by the Final CAIR

Pollutant/Effect	Quantified and Monetized in Base Estimates ^a	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects - Changes in
PM/Health ^d	 Premature mortality based on cohort study estimates^e Bronchitis: chronic and acute Hospital admissions: respiratory and cardiovascular Emergency room visits for asthma Nonfatal heart attacks (myocardial infarction) Lower and upper respiratory illness Minor restricted-activity days Work loss days Asthma exacerbations (asthmatic population) Respiratory symptoms (asthmatic population) Infant mortality 	Premature mortality: short term exposures ^f Subchronic bronchitis cases	Low birth weight Pulmonary function Chronic respiratory diseases other than chronic bronchitis Nonasthma respiratory emergency room visits UVb exposure (+/-) ^g
PM/Welfare	Visibility in Southeastern Class I areas	Visibility in northeastern and Midwestern Class I areas Household soiling	Visibility in western U.S. Class I areas Visibility in residential and non-Class I areas UVb exposure (+/-) ^g

Table 4-2. Human Health and Welfare Effects of Pollutants Affected by the Final CAIR (continued)

Pollutant/Effect	Quantified and Monetized in Base Estimates ^a	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects - Changes in:
Nitrogen and Sulfate			Commercial forests due to acidic sulfate and nitrate deposition
Deposition/ Welfare			Commercial freshwater fishing due to acidic deposition
			Recreation in terrestrial ecosystems due to acidic deposition
			Commercial fishing, agriculture, and forests due to nitrogen deposition
			Recreation in estuarine ecosystems due to nitrogen deposition
			Ecosystem functions
			Passive fertilization
SO ₂ /Health			Hospital admissions for respiratory and cardiac diseases
			Respiratory symptoms in asthmatics
NO _x /Health			Lung irritation
			Lowered resistance to respiratory infection
			Hospital admissions for respiratory and cardiac diseases

Table 4-2. Human Health and Welfare Effects of Pollutants Affected by the Final CAIR (continued)

Table 4-2.	Human Hea	lth and Welfar	e Effects of	f Pollutants .	Affected by the	e Final CAIR (continued)

Pollutant/Effect	Quantified and Monetized in Base Estimates ^a	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
Mercury Health			Incidences of neurological disorders
			Incidences of learning disabilities
			Incidences in developmental delays
			Potential cardiovascular effects ^h , including:
			- Altered blood pressure regulation ^h
			- Increased heart rate variability ^h
			- Incidences of Myocardial infarction ^h
			Potential reproductive effects ^h
Mercury			Impact on birds and mammals (e.g., reproductive effects)
Deposition			Impacts to commercial, subsistence, and recreational
Welfare			fishing

Primary quantified and monetized effects are those included when determining the primary estimate of total monetized benefits of CAIR. See Appendix C for a more complete discussion of the benefit estimates.

^b In addition to primary economic endpoints, there are a number of biological responses that have been associated with ozone health including increased airway responsiveness to stimuli, inflammation in the lung, acute inflammation and respiratory cell damage, and increased susceptibility to respiratory infection. The public health impact of these biological responses may be partly represented by our quantified endpoints.

^c Premature mortality associated with ozone is not currently included in the primary analysis. Recent evidence suggests that short-term exposures to ozone may have a significant effect on daily mortality rates, independent of exposure to PM. EPA is currently conducting a series of meta-analyses of the ozone mortality epidemiology literature. EPA will consider including ozone mortality in primary benefits analyses once a peer-reviewed methodology is available.

^d In addition to primary economic endpoints, there are a number of biological responses that have been associated with PM health effects including morphological changes and altered host defense mechanisms. The public health impact of these biological responses may be partly represented by our quantified endpoints.

^e Cohort estimates are designed to examine the effects of long term exposures to ambient pollution, but relative risk estimates may also incorporate some effects due to shorter term exposures (see Kunzli, 2001 for a discussion of this issue).

^f While some of the effects of short term exposure are likely to be captured by the cohort estimates, there may be additional premature mortality from short term PM exposure not captured in the cohort estimates included in the primary analysis.

^g May result in benefits or disbenefits. See Section 5.3.4 for more details.

^h These are potential effects as the literature is insufficient.

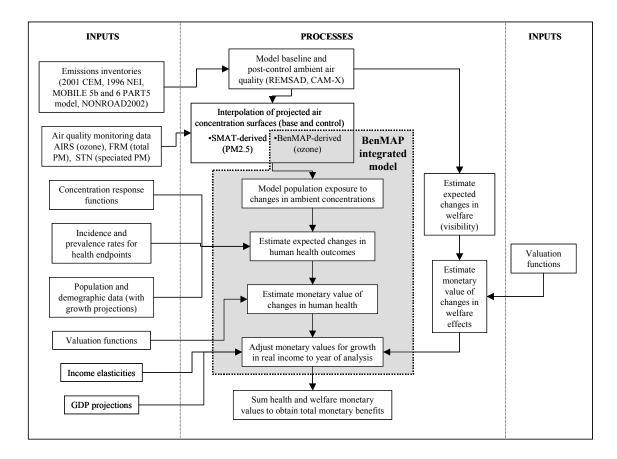


Figure 4-1. Key Steps in Air Quality Modeling Based Benefits Analysis

monitoring data to estimate population-level potential exposures to changes in ambient concentrations for use in estimating health effects. Modeled changes in ambient data are also used to estimate changes in visibility and changes in other air quality statistics that are necessary to estimate welfare effects. Changes in population exposure to ambient air pollution are then input to impact functions² to generate changes in the incidence of health

²The term "impact function" as used here refers to the combination of a) an effect estimate obtained from the epidemiological literature, b) the baseline incidence estimate for the health effect of interest in the modeled population, c) the size of that modeled population, and d) the change in the ambient air pollution metric of interest. These elements are combined in the impact function to generate estimates of changes in incidence of the health effect. The impact function is distinct from the C-R function, which strictly refers to the estimated equation from the epidemiological study relating incidence of the health effect and ambient pollution. We refer to the specific value of the relative risk or estimated coefficients in the epidemiological study as the "effect estimate." In referencing the functions used to generate changes in incidence of health effects for this RIA, we use the term "impact function" rather than C-R function because "impact function" includes all key input parameters used in the incidence calculation.

effects, or changes in other exposure metrics are input to dose-response functions to generate changes in welfare effects. The resulting effects changes are then assigned monetary values, taking into account adjustments to values for growth in real income out to the year of analysis (values for health and welfare effects are in general positively related to real income levels). Finally, values for individual health and welfare effects are summed to obtain an estimate of the total monetary value of the changes in emissions.

The benefits discussed in this chapter represent the estimates based upon emission changes anticipated for the final CAIR program (final CAIR plus the proposal to include SO_2 and annual NO_x controls for New Jersey and Delaware in the final CAIR program) with one exception. The benefits estimated in this report are slightly overstated due to the inclusion of emission reductions for SO_2 and annual NO_x controls for Arkansas. Thus, the analysis presented reflects the EPA's best estimate of the benefits for a complete CAIR program assuming New Jersey and Delaware become a part of the CAIR region for $PM_{2.5}$ as well as ozone, but these benefits are slightly overstated due to use of modeling that includes Arkansas in the CAIR region for SO_2 and annual NO_x controls.

On September 26, 2002, the National Research Council (NRC) released a report on its review of the Agency's methodology for analyzing the health benefits of measures taken to reduce air pollution. The report focused on EPA's approach for estimating the health benefits of regulations designed to reduce concentrations of ambient PM.

In its report, the NRC said that EPA has generally used a reasonable framework for analyzing the health benefits of PM-control measures. It recommended, however, that the Agency take a number of steps to improve its benefits analysis. The current analysis reflects the following suggestions of that NRC report:

- estimate benefits for intervals, such as every 5 years, rather than a single year;
- clearly state the projected baseline statistics used in estimating health benefits, including those for air emissions, air quality, and health outcomes;
- when appropriate, use data from non-U.S. studies to broaden age ranges to which current estimates apply and to include more types of relevant health outcomes;

In addition, the NRC recommended that EPA move the assessment of uncertainties from its ancillary analyses into its base analyses by conducting probabilistic, multiple-source uncertainty analyses. However, for this rule, given the limited data available for such a complex uncertainty assessment, EPA made the decision only to summarize the results of an ancillary probabilistic uncertainty analysis to provide context for sources of uncertainty reflecting statistical error in the base analysis. The EPA followed the NRC recommendations that the probabilistic assessment should be based on available data and expert judgment.

The NRC made a number of recommendations for improving EPA's approach and found that the studies selected by the Agency for use in its benefits analyses were generally reasonable choices. In particular, the NRC agreed with EPA's decision to use cohort studies for estimating premature mortality benefits. It also concluded that the Agency's selection of the American Cancer Society (ACS) study for the evaluation of PM-related premature mortality was reasonable, although it noted the publication of new cohort studies that the Agency should evaluate. Since the publication of the NRC report, EPA has reviewed new cohort studies, including reanalyses of the ACS study data and has carefully considered these new study data in developing the analytical approach for CAIR (see below).

In addition to the NRC report, EPA received technical guidance and input regarding its methodology for conducting PM- and ozone-related benefits analysis from the Health Effects Subgroup (HES) of the SAB Council reviewing the 812 blueprint (SAB-HES, 2004) and the Office of Management and Budget (OMB) through ongoing discussions regarding methods used in conducting regulatory impact analyses (RIAs), and developments during the collaboration on the recent Nonroad Diesel rulemaking. EPA addressed many of the comments received from the NRC, the SAB-HES, and OMB in developing the analytical approach for the recent Nonroad Diesel Rule RIA. These improvements are also reflected in this analysis for the final CAIR.

Recommendations from OMB regarding RIA methods have focused on the approach used to characterize uncertainty in the benefits estimates generated for RIAs and the approach used to value mortality estimates. EPA is currently developing a comprehensive integrated strategy for characterizing the impact of uncertainty in key elements of the benefits modeling process (e.g., emissions modeling, air quality modeling, health effects incidence estimation, valuation) on the health impact and monetized benefits estimates that are generated. A subset of this effort, which has recently been completed and peer reviewed, was a pilot expert elicitation designed to characterize uncertainty in the estimation of PM- related mortality resulting from both short-term and long-term exposure.³ The peer review of the pilot expert elicitation was generally favorable. We provide a detailed description of the pilot in Appendix B, along with a summary of results in Section 4.3.

We have also reflected advances in data and methods in air quality modeling, epidemiology, and economics in developing this analysis. Updates to the assumptions and methods used in estimating $PM_{2.5}$ -related and ozone-related benefits since the analysis for the proposed rule include the following:

Air Quality

- Use of CMAQ-based predictions for ambient PM_{2.5} and component species.
- Use of an updated SMAT approach for developing PM_{2.5} air modeling results. For the CAIR proposal analysis, we used temporally scaled speciated PM_{2.5} monitor data from 2001–2002, reconstructed into total PM_{2.5} mass based on 2000–2002 design values and kriged to 12 kilometer grids (nested within the standard 36 km REMSAD grid structure). Temporal scaling was based on ratios of future modeled REMSAD data to 2001 REMSAD model data, using REMSAD modeling conducted at the 36 km grid resolution. For this analysis of the final rule, we used a modified method that is based on the future to 2001 modeled CMAQ speciated outputs and spatially interpolated speciated monitor data (see Chapter 3 for more details).
- The CAIR proposal analysis was limited to the Eastern U.S. For the final rule analysis, PM benefits are estimated for the entire U.S., to account for the transport of PM precursor emissions from the CAIR domain to the western states. The ozone benefits assessment is still limited to the eastern U.S. due to limitations in the models for ozone formation in the western states.

Valuation

• In generating the monetized benefits for reductions in premature mortality in the primary analysis, a 20-year segmented lag structure will be used to characterize the relationship between the time when exposure to ambient PM_{2.5} is changed and the time when reductions in premature mortality are expected to occur.

³Expert elicitation is a formal, highly structured and well documented process whereby expert judgments, usually of multiple experts, are obtained (Ayyub, 2002).

Uncertainty

• In addition to the primary estimate of the benefits of reduced premature mortality, we characterize uncertainty using a probabilistic range of benefits based on statistical uncertainty as captured in the standard errors associated with the Pope et al (2002) epidemiological study, and model uncertainties obtained from the pilot expert elicitation. Uncertainty in some other elements of the model are characterized by statistical uncertainty as captured in either standard errors on epidemiological effect estimates or variability in published estimates of valuation estimates.

The benefits estimates generated for the final CAIR are subject to a number of assumptions and uncertainties, which are discussed throughout this document. For example, key assumptions underlying the primary estimate for the mortality category include the following:

- Inhalation of fine particles is causally associated with premature death at concentrations near those experienced by most Americans on a daily basis. Although biological mechanisms for this effect have not yet been completely established, the weight of the available epidemiological and experimental evidence supports an assumption of causality.
- 2) All fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because PM produced via transported precursors emitted from EGUs may differ significantly from direct PM released from automotive engines and other industrial sources. However, no clear scientific grounds exist for supporting differential effects estimates by particle type.
- 3) The C-R function for fine particles is approximately linear within the range of ambient concentrations under consideration. Thus, the estimates include health benefits from reducing fine particles in areas with varied concentrations of PM, including both regions that are in attainment with the fine particle standard and those that do not meet the standard.
- 4) The forecasts for future emissions and associated air quality modeling are valid. Although recognizing the difficulties, assumptions, and inherent uncertainties in the overall enterprise, these analyses are based on peer-reviewed scientific literature and up-to-date assessment tools, and we believe the results are highly useful in assessing this rule.

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In addition to the quantified and monetized benefits summarized above, a number of additional categories are not currently amenable to quantification or valuation. These include reduced acid and particulate deposition damage to cultural monuments and other materials, reduced ozone effects on forested ecosystems, and environmental benefits due to reductions of impacts of acidification in lakes and streams and eutrophication in coastal areas. Additionally, we have not quantified a number of known or suspected health effects linked with PM and ozone for which appropriate health impact functions are not available or which do not provide easily interpretable outcomes (i.e., changes in forced expiratory volume [FEV1]). As a result, monetized benefits generated for the primary estimate may underestimate the total benefits attributable to the final regulatory option.

Benefits estimates for the final CAIR were generated using BenMAP, a computer program developed by EPA that integrates a number of the modeling elements used in previous RIAs (e.g., interpolation functions, population projections, health impact functions, valuation functions, analysis and pooling methods) to translate modeled air concentration estimates into health effects incidence estimates and monetized benefits estimates. BenMAP provides estimates of both the mean impacts and the distribution of impacts (more information on BenMAP can be found at http://www.epa.gov/ttn/ecas/ benmodels.html).

In general, this chapter is organized around the steps illustrated in Figure 4-1. In Section 4.1, we provide an overview of the data and methods that were used to quantify and value health and welfare endpoints and discuss how we incorporate uncertainty into our analysis. In Section 4.2, we report the results of the analysis for human health and welfare effects (the overall benefits estimated for the final CAIR are summarized in Table 4-1). Details on the emissions inventory and air modeling are presented in Chapter 3.

4.1 Benefit Analysis—Data and Methods

Given changes in environmental quality (ambient air quality, visibility, nitrogen, and sulfate deposition), the next step is to determine the economic value of those changes. We follow a "damage-function" approach in calculating total benefits of the modeled changes in environmental quality. This approach estimates changes in individual health and welfare endpoints (specific effects that can be associated with changes in air quality) and assigns values to those changes assuming independence of the individual values. Total benefits are calculated simply as the sum of the values for all nonoverlapping health and welfare endpoints. This imposes no overall preference structure and does not account for potential income or substitution effects (i.e., adding a new endpoint will not reduce the value of changes in other endpoints). The "damage-function" approach is the standard approach for

most cost-benefit analyses of environmental quality programs and has been used in several recent published analyses (Banzhaf et al., 2002; Levy et al., 2001; Levy et al., 1999; Ostro and Chestnut, 1998).

To assess economic value in a damage-function framework, the changes in environmental quality must be translated into effects on people or on the things that people value. In some cases, the changes in environmental quality can be directly valued, as is the case for changes in visibility. In other cases, such as for changes in ozone and PM, a health and welfare impact analysis must first be conducted to convert air quality changes into effects that can be assigned dollar values.

For the purposes of this RIA, the health impacts analysis is limited to those health effects that are directly linked to ambient levels of air pollution and specifically to those linked to ozone and PM. There may be other, indirect health impacts associated with implementing emissions controls, such as occupational health impacts for coal miners. These impacts may be positive or negative, but in general, for this set of control options, they are expected to be small relative to the direct air pollution-related impacts.

The welfare impacts analysis is limited to changes in the environment that have a direct impact on human welfare. For this analysis, we are limited by the available data to examine impacts of changes in visibility. We also provide qualitative discussions of the impact of changes in other environmental and ecological effects, for example, changes in deposition of nitrogen and sulfur to terrestrial and aquatic ecosystems, but we are unable to place an economic value on these changes.

We note at the outset that EPA rarely has the time or resources to perform extensive new research to measure either the health outcomes or their values for this analysis. Thus, similar to Kunzli et al. (2000) and other recent health impact analyses, our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Adjustments are made for the level of environmental quality change, the sociodemographic and economic characteristics of the affected population, and other factors to improve the accuracy and robustness of benefits estimates.

4.1.1 Valuation Concepts

In valuing health impacts, we note that reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects by a fairly small amount for a large population. The appropriate economic measure is willingness to pay⁴ (WTP) for changes in risk prior to the regulation (Freeman, 1993).⁵ Adoption of WTP as the measure of value implies that the value of environmental quality improvements depends on the individual preferences of the affected population and that the existing distribution of income (ability to pay) is appropriate. For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as the measure of benefits. These cost of illness (COI) estimates generally understate the true value of reductions in risk of a health effect, because they do not include the value of avoided pain and suffering from the health effect (Harrington and Portney, 1987; Berger et al., 1987).

One distinction in environmental benefits estimation is between use values and nonuse values. Although no general agreement exists among economists on a precise distinction between the two (see Freeman [1993]), the general nature of the difference is clear. Use values are those aspects of environmental quality that affect an individual's welfare directly. These effects include changes in product prices, quality, and availability; changes in the quality of outdoor recreation and outdoor aesthetics; changes in health or life expectancy; and the costs of actions taken to avoid negative effects of environmental quality changes.

Nonuse values are those for which an individual is willing to pay for reasons that do not relate to the direct use or enjoyment of any environmental benefit but might relate to existence values and bequest values. Nonuse values are not traded, directly or indirectly, in markets. For this reason, measuring nonuse values has proven to be significantly more

⁴For many goods, WTP can be observed by examining actual market transactions. For example, if a gallon of bottled drinking water sells for \$1, it can be observed that at least some people are willing to pay \$1 for such water. For goods not exchanged in the market, such as most environmental "goods," valuation is not as straightforward. Nevertheless, a value may be inferred from observed behavior, such as sales and prices of products that result in similar effects or risk reductions (e.g., nontoxic cleaners or bike helmets). Alternatively, surveys can be used in an attempt to directly elicit WTP for an environmental improvement.

⁵In general, economists tend to view an individual's WTP for an improvement in environmental quality as the appropriate measure of the value of a risk reduction. An individual's willingness to accept (WTA) compensation for not receiving the improvement is also a valid measure. However, WTP is generally considered to be a more readily available and conservative measure of benefits.

difficult than measuring use values. The air quality changes produced by CAIR cause changes in both use and nonuse values, but the monetary benefits estimates are almost exclusively for use values.

More frequently than not, the economic benefits from environmental quality changes are not traded in markets, so direct measurement techniques cannot be used. There are three main nonmarket valuation methods used to develop values for endpoints considered in this analysis: stated preference (or contingent valuation [CV]), indirect market (e.g., hedonic wage), and avoided cost methods.

The stated preference or CV method values endpoints by using carefully structured surveys to ask a sample of people what amount of compensation is equivalent to a given change in environmental quality. There is an extensive scientific literature and body of practice on both the theory and technique of stated preference-based valuation. Well-designed and well-executed stated preference studies are valid for estimating the benefits of air quality regulations.⁶ Stated preference valuation studies form the basis for valuing a number of health and welfare endpoints, including the value of mortality risk reductions, CB risk reductions, minor illness risk reductions, and visibility improvements.

Indirect market methods can also be used to infer the benefits of pollution reduction. The most important application of this technique for our analysis is the calculation of the VSL for use in estimating benefits from mortality risk reductions. No market exists where changes in the probability of death are directly exchanged. However, people make decisions about occupation, precautionary behavior, and other activities associated with changes in the risk of death. By examining these risk changes and the other characteristics of people's choices, it is possible to infer information about the monetary values associated with changes in mortality risk (see Section 4.1.5).

⁶Concerns about the reliability of value estimates from CV studies arose because research has shown that bias can be introduced easily into these studies if they are not carefully conducted. Accurately measuring WTP for avoided health and welfare losses depends on the reliability and validity of the data collected. There are several issues to consider when evaluating study quality, including but not limited to 1) whether the sample estimates of WTP are representative of the population WTP; 2) whether the good to be valued is comprehended and accepted by the respondent; 3) whether the WTP elicitation format is designed to minimize strategic responses; 4) whether WTP is sensitive to respondent familiarity with the good, to the size of the change in the good, and to income; 5) whether the estimates of WTP are broadly consistent with other estimates of WTP for similar goods; and 6) the extent to which WTP responses are consistent with established economic principles.

Avoided cost methods are ways to estimate the costs of pollution by using the expenditures made necessary by pollution damage. For example, if buildings must be cleaned or painted more frequently as levels of PM increase, then the appropriately calculated increment of these costs is a reasonable lower-bound estimate (under most conditions) of true economic benefits when PM levels are reduced. Avoided costs methods are also used to estimate some of the health-related benefits related to morbidity, such as hospital admissions (see Section 4.1.5).

4.1.2 Growth in WTP Reflecting National Income Growth Over Time

Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. There is substantial empirical evidence that the income elasticity⁷ of WTP for health risk reductions is positive, although there is uncertainty about its exact value. Thus, as real income increases, the WTP for environmental improvements also increases. Although many analyses assume that the income elasticity of WTP is unit elastic (i.e., a 10 percent higher real income level implies a 10 percent higher WTP to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefits estimates in two different ways: through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and through differences in income between study populations and the affected populations at a particular time. Empirical evidence of the effect of real income on WTP gathered to date is based on studies examining the former. The Environmental Economics Advisory Committee (EEAC) of the Science Advisory Board (SAB) advised EPA to adjust WTP for increases in real income over time but not to adjust WTP to account for cross-sectional income differences "because of the sensitivity of making such distinctions, and because of insufficient evidence available at present" (EPA-SAB-EEAC-00-013). A recent advisory by another committee associated with the SAB, the Advisory Council on Clean Air Compliance Analysis, has provided conflicting advice. While agreeing with "the general principle that the willingness to pay to reduce mortality risks is likely to increase with growth in real income. The same increase

⁷Income elasticity is a common economic measure equal to the percentage change in WTP for a 1 percent change in income.

should be assumed for the WTP for serious nonfatal health effects (EPA-SAB-COUNCIL-ADV-04-004, p. 52)," they note that "given the limitations and uncertainties in the available empirical evidence, the Council does not support the use of the proposed adjustments for aggregate income growth as part of the primary analysis (EPA-SAB-COUNCIL-ADV-04-004, p. 53)." Until these conflicting advisories have been reconciled, EPA will continue to adjust valuation estimates to reflect income growth using the methods described below.

Based on a review of the available income elasticity literature, we adjusted the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, we applied estimates derived from cross-sectional studies in our analysis. Details of the procedure can be found in Kleckner and Neumann (1999). An abbreviated description of the procedure we used to account for WTP for real income growth between 1990 and 2010 and 2015 is presented below.

Reported income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP. As such, we use different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. Note that because of the variety of empirical sources used in deriving the income elasticities, there may appear to be inconsistencies in the magnitudes of the income elasticities relative to the severity of the effects (apriori one might expect that more severe outcomes would show less income elasticity of WTP). We have not imposed any additional restrictions on the empirical estimates of income elasticity. We also expect that the WTP for improved visibility in Class I areas would increase with growth in real income. The relative magnitude of the income elasticity of WTP for visibility compared with those for health effects suggests that visibility is not as much of a necessity as health, thus, WTP is more elastic with respect to income. The elasticity values used to adjust estimates of benefits in 2010 and 2015 are presented in Table 4-3.

In addition to elasticity estimates, projections of real gross domestic product (GDP) and populations from 1990 to 2010 and 2015 are needed to adjust benefits to reflect real per capita income growth. For consistency with the emissions and benefits modeling, we used national population estimates for the years 1990 to 1999 based on U.S. Census Bureau estimates (Hollman et al., 2000). These population estimates are based on application of a

Table 4-3.	Elasticity	Values	Used to	Account for	Projected	Real Income Growth ^a	ı

Benefit Category	Central Elasticity Estimate
Minor Health Effect	0.14
Severe and Chronic Health Effects	0.45
Premature mortality	0.40
Visibility	0.90

^a Derivation of estimates can be found in Kleckner and Neumann (1999) and Chestnut (1997). COI estimates are assigned an adjustment factor of 1.0.

cohort-component model applied to 1990 U.S. Census data projections (U.S. Bureau of Census, 2000). For the years between 2000 and 2015, we applied growth rates based on the U.S. Census Bureau projections to the U.S. Census estimate of national population in 2000. We used projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010.⁸ We used projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's (2000) for the years 2010 to 2015.⁹

Using the method outlined in Kleckner and Neumann (1999) and the population and income data described above, we calculated WTP adjustment factors for each of the elasticity estimates listed in Table 4-4. Benefits for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility) are adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor. Table 4-4 lists the estimated adjustment factors. Note that, for premature mortality, we applied the income adjustment factor to the present discounted value of the stream of avoided mortalities occurring over the lag period. Also note that because of a lack of data on the dependence of COI and income, and a lack of data on projected growth in average wages, no adjustments are made to benefits based on the COI approach or to work loss days and worker productivity. This assumption leads us to underpredict benefits in future years because it is

⁸U.S. Bureau of Economic Analysis, Table 2A (1992\$) (available at http://www.bea.doc.gov/bea/dn/0897nip2/ tab2a.htm.) and U.S. Bureau of Economic Analysis, Economics and Budget Outlook. Note that projections for 2007 to 2010 are based on average GDP growth rates between 1999 and 2007.

⁹In previous analyses, we used the Standard and Poor's projections of GDP directly. This led to an apparent discontinuity in the adjustment factors between 2010 and 2011. We refined the method by applying the relative growth rates for GDP derived from the Standard and Poor's projections to the 2010 projected GDP based on the Bureau of Economic Analysis projections.

Benefit Category	2010	2015
Minor Health Effect	1.034	1.073
Severe and Chronic Health Effects	1.113	1.254
Premature Mortality	1.100	1.222
Visibility	1.239	1.581

Table 4-4. Adjustment Factors Used to Account for Projected Real Income Growth^a

^a Based on elasticity values reported in Table 4-3, U.S. Census population projections, and projections of real GDP per capita.

likely that increases in real U.S. income would also result in increased COI (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher).

4.1.3 Methods for Describing Uncertainty

In any complex analysis using estimated parameters and inputs from numerous models, there are likely to be many sources of uncertainty. This analysis is no exception. As outlined both in this and preceding chapters, many inputs were used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological health effect estimates, estimates of values (both from WTP and COI studies), population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Each of these inputs may be uncertain and, depending on its role in the benefits analysis, may have a disproportionately large impact on final estimates of total benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis. When compounded with uncertainty in later stages, small uncertainties in emission levels can lead to large impacts on total benefits.

Some key sources of uncertainty in each stage of the benefits analysis are the following:

- gaps in scientific data and inquiry;
- variability in estimated relationships, such as epidemiological effect estimates, introduced through differences in study design and statistical modeling;

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- errors in measurement and projection for variables such as population growth rates;
- errors due to misspecification of model structures, including the use of surrogate variables, such as using PM₁₀ when PM_{2.5} is not available, excluded variables, and simplification of complex functions; and
- biases due to omissions or other research limitations.

Some of the key uncertainties in the benefits analysis are presented in Table 4-5.

The NRC report on EPA's benefits analysis methodology highlighted the need for EPA to conduct rigorous quantitative analysis of uncertainty in its benefits estimates. In response to these comments, EPA has initiated the development of a comprehensive methodology for characterizing the aggregate impact of uncertainty in key modeling elements on both health incidence and benefits estimates. For this analysis of the final CAIR, EPA has developed a limited probabilistic simulation approach based on Monte Carlo methods to propagate the impact of a limited set of sources of uncertainty through the modeling framework. Issues such as correlation between input parameters and the identification of reasonable upper and lower bounds for input distributions characterizing uncertainty in additional model elements will be addressed in future versions of the uncertainty framework.

One component of EPA's uncertainty analysis methodology that is partially reflected in the CAIR analysis is our work using the results of an expert elicitation to characterize uncertainty in the effect estimates used to estimate premature mortality resulting from both short-term and longer-term exposures to PM. This expert elicitation was aimed at evaluating uncertainty in both the form of the mortality impact function (e.g., threshold versus linear models) and the fit of a specific model to the data (e.g., confidence bounds for specific percentiles of the mortality effect estimates). Additional issues, such as the ability of longterm cohort studies to capture premature mortality resulting from short-term peak PM exposures, were also addressed in the expert elicitation. In collaboration with OMB, EPA completed a pilot expert elicitation which is used in the ancillary uncertainty analysis for CAIR (as discussed in Section 4.3). Based on our experience during the pilot, EPA plans to conduct a full-scale expert elicitation in 2005 that will provide a more robust characterization of the uncertainty in the premature mortality function.

Table 4-5. Primary Sources of Uncertainty in the Benefits Analysis

- 1. Uncertainties Associated with Impact Functions
- The value of the ozone or PM effect estimate in each impact function.
- Application of a single impact function to pollutant changes and populations in all locations.
- Similarity of future-year impact functions to current impact functions.
- Correct functional form of each impact function.
- Extrapolation of effect estimates beyond the range of ozone or PM concentrations observed in the source epidemiological study.
- Application of impact functions only to those subpopulations matching the original study population.
- 2. Uncertainties Associated with Ozone and PM Concentrations
- Responsiveness of the models to changes in precursor emissions resulting from the control policy.
- Projections of future levels of precursor emissions, especially ammonia and crustal materials.
- Model chemistry for the formation of ambient nitrate concentrations.
- Lack of ozone monitors in rural areas requires extrapolation of observed ozone data from urban to rural areas.
- Use of separate air quality models for ozone and PM does not allow for a fully integrated analysis of pollutants and their interactions.
- Full ozone season air quality distributions are extrapolated from a limited number of simulation days.
- Comparison of model predictions of particulate nitrate with observed rural monitored nitrate levels indicates that REMSAD overpredicts nitrate in some parts of the Eastern United States
- 3. Uncertainties Associated with PM Mortality Risk
- Limited scientific literature supporting a direct biological mechanism for observed epidemiological evidence.
- Direct causal agents within the complex mixture of PM have not been identified.
- The extent to which adverse health effects are associated with low-level exposures that occur many times in the year versus peak exposures.
- The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than
 the levels occurring during the period of study.
- Reliability of the limited ambient PM_{2.5} monitoring data in reflecting actual PM_{2.5} exposures.
- 4. Uncertainties Associated with Possible Lagged Effects
- The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels that would occur in a single year is uncertain as well as the portion that might occur in subsequent years.
- 5. Uncertainties Associated with Baseline Incidence Rates
- Some baseline incidence rates are not location specific (e.g., those taken from studies) and therefore may not accurately represent the
 actual location-specific rates.
- Current baseline incidence rates may not approximate well baseline incidence rates in 2015.
- Projected population and demographics may not represent well future-year population and demographics.
- 6. Uncertainties Associated with Economic Valuation
- Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them.
- Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates because of differences in income or other factors.
- 7. Uncertainties Associated with Aggregation of Monetized Benefits
- Health and welfare benefits estimates are limited to the available impact functions. Thus, unquantified or unmonetized benefits are not included.

For the final CAIR, EPA addressed key sources of uncertainty through Monte Carlo propagation of uncertainty in the C-R functions and economic valuation functions and through a series of sensitivity analyses examining the impact of alternate assumptions on the benefits estimates that are generated. It should be noted that the Monte Carlo-generated distributions of benefits reflect only some of the uncertainties in the input parameters. Uncertainties associated with emissions, air quality modeling, populations, and baseline health effect incidence rates are not represented in the distributions of benefits for CAIR.

Our point estimate of total benefits is uncertain because of the uncertainty in model elements discussed above (see Table 4-5). Uncertainty about specific aspects of the health and welfare estimation models is discussed in greater detail in the following sections. The total benefits estimate may understate or overstate actual benefits of the rule.

In considering the monetized benefits estimates, the reader should remain aware of the many limitations of conducting the analyses mentioned throughout this RIA. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the effects listed in Table 4-1. For many health and welfare effects, such as changes in ecosystem functions and PM-related materials damage, reliable impact functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefit categories, the benefits estimates presented in this analysis would increase, although the magnitude of such an increase is highly uncertain. Unquantified benefits are qualitatively discussed in the health and welfare effects sections. Furthermore, EPA explores the implication of the potential relationship between O₃ and premature mortality in its sensitivity analysis. In addition to unquantified benefits, there may also be environmental costs (disbenefits) that we are unable to quantify. These endpoints are qualitatively discussed in the health and welfare effects sections as well. The net effect of excluding benefit and disbenefit categories from the estimate of total benefits depends on the relative magnitude of the effects.

Although we are not currently able to estimate the magnitude of these unquantified and unmonetized benefits, specific categories merit further discussion. The EPA believes there is considerable value to the public for the benefit categories that could not be monetized. With regard to unmonetized PM-related health benefit categories listed in Table 4-2, we feel these benefits may be small relative to those categories we were able to quantify and monetize.

However, there is one category where new studies suggest the possibility of significant additional economic benefits. Over the past several years, EPA's SAB has

expressed the view that there were not sufficient data to show a separate ozone mortality effect, in essence, saying that any ozone benefits are captured in the PM-related mortality benefit estimates. However, in their most recent advice, the SAB recommended that EPA reconsider the evidence on ozone-related mortality based on the publication of several recent analyses that found statistically significant associations between ozone and mortality. Based on these studies and the recommendations from the SAB, EPA has sponsored three independent meta-analyses of the ozone-mortality epidemiology literature to inform a determination on including this important health endpoint. The studies are complete and have been accepted for publication in the journal Epidemiology in July 2005 [see Bell et al., in press; Ito et al., in press; Levy et al., in press].

The Agency believes that publication of these meta-analyses will significantly enhance the scientific defensibility of benefits estimates for ozone, that include the benefits of premature mortality reductions. In addition, a study published in JAMA in November 2004 also confirmed that ozone mortality impacts can be calculated separately from PM mortality impacts (Bell et al., 2004). EPA's believes that there is sufficient evidence to return to the SAB to confirm that these studies address their previous concerns. Using effect estimates similar to those found in these new studies, EPA estimates the monetary value of the ozone-related premature mortality benefits could be substantial. We estimate ozone mortality benefits may yield roughly 500 reduced premature mortalities per year and may increase the benefits of CAIR by approximately \$3 billion annually.

In addition to unquantified and unmonetized health benefit categories, Table 4-2 shows a number of welfare benefit categories that are omitted from the monetized benefit estimates for this rule. Only a subset of the expected visibility benefits-those for Class I areas in the southeastern United States are included in the monetary benefits estimates we project for this rule. We believe the benefits associated with these non-health benefit categories are likely significant. For example, we are able to quantify significant visibility improvements in Class I areas in the Northeast and Midwest, but are unable at present to place a monetary value on these improvement. Similarly, we anticipate improvement in visibility in residential areas within the CAIR region for which we are currently unable to monetize benefits. For the Class I areas in the southeastern U.S., we estimate annual benefits of \$1.78 billion beginning in 2015 for visibility improvements. The value of visibility benefits in areas where we were unable to monetize benefits could also be substantial.

We conduct supplemental analyses related to visibility and household cleaning costs later in this chapter. Based on these analyses, expanded coverage of these benefit categories could increase total benefits by over \$500 million. (See Appendix C for more details.)

In a recent study, Resources for the Future (RFF) estimates total benefits (i.e., the sum of use and nonuse values) of natural resource improvements for the Adirondacks resulting from a program that would reduce acidification in 40 percent of the lakes in the Adirondacks of concern for acidification (Banzhaf, 2004). While the study requires further evaluation, the RFF study does suggests that the benefits of acid deposition reductions for CAIR could be substantial in terms of the total monetized value for ecological endpoints.

Another area of potential benefits not monetized relates to potential reductions in nitrogen deposition from CAIR for estuaries and coastal waters within the CAIR region. Nitrogen deposition contributes to eutrophication and water quality degradation in estuaries and coastal waters. While we are unable to monetize the benefits of such reductions, the Chesapeake Bay Program estimated the reduced mass of delivered nitrogen loads likely to result from CAIR, based upon the CAIR proposal deposition estimates published in January 2004. Atmospheric deposition of nitrogen accounts for a significant portion of the nitrogen loads to the Chesapeake with 28 percent of the nitrogen loads to the watershed coming from air deposition. Based upon the CAIR proposal nitrogen deposition rates published in the January 2004 proposal, the Chesapeake Bay Program finds that CAIR will likely reduce the nitrogen loads to the Bay by 10 million pounds per year by 2010 (Sweeney, 2004). These substantial nitrogen load reductions more than fulfill the EPA's commitment to reduce atmospheric deposition delivered to the Chesapeake Bay by 8 million pounds annually. The benefits of these atmospheric deposition reductions for the Bay are likely to be substantial.

4.1.4 Demographic Projections

Quantified and monetized human health impacts depend on the demographic characteristics of the population, including age, location, and income. We use projections based on economic forecasting models developed by Woods and Poole, Inc. The Woods and Poole (WP) database contains county-level projections of population by age, sex, and race out to 2025. Projections in each county are determined simultaneously with every other county in the United States to take into account patterns of economic growth and migration. The sum of growth in county-level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates (Hollman et al., 2000). According to WP, linking county-level growth projections together and constraining to a national-level total growth avoids potential errors introduced by forecasting

each county independently. County projections are developed in a four-stage process. First, national-level variables such as income, employment, and populations are forecasted. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an "export-base" approach, which relies on linking industrial-sector production of nonlocally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-based approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector. Third, population is projected for each economic area based on net migration rates derived from employment opportunities and following a cohort-component method based on fertility and mortality in each area. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2015 based on historical rates of mortality, fertility, and migration.

The WP projections of county-level population are based on historical population data from 1969 through 1999 and do not include the 2000 Census results. Given the availability of detailed 2000 Census data, we constructed adjusted county-level population projections for each future year using a two-stage process. First, we constructed ratios of the projected WP populations in a future year to the projected WP population in 2000 for each future year by age, sex, and race. Second, we multiplied the block-level 2000 Census population data by the appropriate age-, sex-, and race-specific WP ratio for the county containing the census block for each future year. This results in a set of future population projections that is consistent with the most recent detailed Census data.

As noted above, values for environmental quality improvements are expected to increase with growth in real per capita income. Accounting for real income growth over time requires projections of both real GDP and total U.S. populations. For consistency with the emissions and benefits modeling, we used national population estimates based on the U.S. Census Bureau projections.

4.1.5 Health Benefits Assessment Methods

The largest monetized benefits of reducing ambient concentrations of PM and ozone are attributable to reductions in health risks associated with air pollution. EPA's Criteria Documents for ozone and PM list numerous health effects known to be linked to ambient concentrations of these pollutants (EPA, 1996a; 1996b). As illustrated in Figure 4-1, quantification of health impacts requires several inputs, including epidemiological effect

estimates (concentration-response functions), baseline incidence and prevalence rates, potentially affected populations, and estimates of changes in ambient concentrations of air pollution. Previous sections have described the population and air quality inputs. This section describes the effect estimates and baseline incidence and prevalence inputs and the methods used to quantify and monetize changes in the expected number of incidences of various health effects.

4.1.5.1 Selecting Health Endpoints and Epidemiological Effect Estimates

Certain quantified health benefits of the rule may be related to ozone only, PM only, or both pollutants. Based on the available epidemiological data, we quantified decreased worker productivity, respiratory hospital admissions for children under two years of age, and school absences related to ozone but not PM. The PM-only health effects we quantified include premature mortality, nonfatal heart attacks, CB, acute bronchitis, upper and lower respiratory symptoms, asthma exacerbations, and days of work lost. The health effects that we quantified relate to both PM and ozone include hospital admissions, emergency room visits for asthma, and MRADs. Although recent epidemiological evidence points to an association between short term exposures to ozone and premature mortality, EPA is not prepared to quantify this impact in the primary analysis for the CAIR due to the need for additional review of the issue by the Health Effects Subcommittee of the SAB.

We relied on the published scientific literature to ascertain the relationship between PM and ozone exposure and adverse human health effects. We evaluated studies using the selection criteria summarized in Table 4-6. These criteria include consideration of whether the study was peer reviewed, the match between the pollutant studied and the pollutant of interest, the study design and location, and characteristics of the study population, among other considerations. The selection of C-R functions for the benefits analysis is guided by the goal of achieving a balance between comprehensiveness and scientific defensibility.

Some health effects are excluded from this analysis for three reasons: the possibility of double-counting (such as hospital admissions for specific respiratory diseases), uncertainties in applying effect relationships based on clinical studies to the affected population, or a lack of an established relationship between the health effect and pollutant in the published epidemiological literature. An improvement in ambient PM and ozone air quality may reduce the number of incidences within each unquantified effect category that the U.S. population would experience. Although these health effects are believed to be PM or ozone induced, effect estimates are not available for quantifying the benefits associated

Peer-Reviewed Peer-reviewed research is preferred to research that has not undergone the peer-review Research process. Study Type Among studies that consider chronic exposure (e.g., over a year or longer), prospective cohort studies are preferred over cross-sectional studies because they control for important individual-level confounding variables that cannot be controlled for in cross-sectional studies. Study Period Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and lifestyle over time. However, when there are only a few studies available, studies from all years will be included. Population The most technically appropriate measures of benefits would be based on impact Attributes functions that cover the entire sensitive population but allow for heterogeneity across age or other relevant demographic factors. In the absence of effect estimates specific to age, sex, preexisting condition status, or other relevant factors, it may be appropriate to select effect estimates that cover the broadest population to match with the desired outcome of the analysis, which is total national-level health impacts. Study Size Studies examining a relatively large sample are preferred because they generally have more power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population or through repeated observations on a smaller population (e.g., through a symptom diary recorded for a panel of asthmatic children). U.S. studies are more desirable than non-U.S. studies because of potential differences in Study Location pollution characteristics, exposure patterns, medical care system, population behavior, and lifestyle. Pollutants When modeling the effects of ozone and PM (or other pollutant combinations) jointly, it Included in is important to use properly specified impact functions that include both pollutants. Model Using single-pollutant models in cases where both pollutants are expected to affect a health outcome can lead to double-counting when pollutants are correlated. Measure of PM For this analysis, impact functions based on PM25 are preferred to PM10 because CAIR will regulate emissions of $PM_{2.5}$ precursors, and air quality modeling was conducted for this size fraction of PM. Where PM_{2.5} functions are not available, PM₁₀ functions are used as surrogates, recognizing that there will be potential downward (upward) biases if the fine fraction of PM_{10} is more (less) toxic than the coarse fraction. Economically Some health effects, such as forced expiratory volume and other technical measurements Valuable Health of lung function, are difficult to value in monetary terms. These health effects are not Effects quantified in this analysis. Nonoverlapping Although the benefits associated with each individual health endpoint may be analyzed Endpoints separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double-counting of benefits.

Table 4-6. Summary of Considerations Used in Selecting C-R Functions

Comments

Consideration

with reducing these effects.¹⁰ The inability to quantify these effects lends a downward bias to the monetized benefits presented in this analysis.

In general, the use of results from more than a single study can provide a more robust estimate of the relationship between a pollutant and a given health effect. However, there are often differences between studies examining the same endpoint, making it difficult to pool the results in a consistent manner. For example, studies may examine different pollutants or different age groups. For this reason, we consider very carefully the set of studies available examining each endpoint and select a consistent subset that provides a good balance of population coverage and match with the pollutant of interest. In many cases, either because of a lack of multiple studies, consistency problems, or clear superiority in the quality or comprehensiveness of one study over others, a single published study is selected as the basis of the effect estimate.

When several effect estimates for a pollutant and a given health endpoint have been selected, they are quantitatively combined or pooled to derive a more robust estimate of the relationship. The Benefits TSD completed for the nonroad diesel rulemaking provides details of the procedures used to combine multiple impact functions (Abt Associates, 2003). In general, we used fixed or random effects models to pool estimates from different studies of the same endpoint. Fixed effects pooling simply weights each study's estimate by the inverse variance, giving more weight to studies with greater statistical power (lower variance). Random effects pooling accounts for both within-study variance and between-study variability, due, for example, to differences in population susceptibility. We used the fixed effects model as our null hypothesis and then determined whether the data suggest that we should reject this null hypothesis, in which case we would use the random effects model.¹¹ Pooled impact functions are used to estimate hospital admissions (PM), school absence days (ozone), lower respiratory symptoms (PM), asthma exacerbations (PM), and asthma-related emergency room visits (ozone). For more details on methods used to pool

¹⁰There has been a great deal of research recently on the potential effect of ozone on premature mortality (Anderson et al, 2004; Bell et al, 2004; Thurston and Ito, 2001). Although the air pollutant most clearly associated with premature mortality is PM, with dozens of studies reporting such an association, repeated ozone exposure is a likely contributing factor for premature mortality, causing an inflammatory response in the lungs that may predispose elderly and other sensitive individuals to become more susceptible. Appendix C presents a sensitivity analysis showing the potential impacts of CAIR on ozone-related mortality.

¹¹In this analysis, the fixed effects model assumes that there is only one pollutant coefficient for the entire modeled area. The random effects model assumes that studies conducted in different locations are estimating different parameters; therefore, there may be a number of different underlying pollutant coefficients.

incidence estimates, see the Benefits TSD for the nonroad diesel rulemaking (Abt Associates, 2003).

Effect estimates for a pollutant and a given health endpoint were applied consistently across all locations nationwide. This applies to both impact functions defined by a single effect estimate and those defined by a pooling of multiple effect estimates. Although the effect estimate may, in fact, vary from one location to another (e.g., because of differences in population susceptibilities or differences in the composition of PM), location-specific effect estimates are generally not available.

The specific studies from which effect estimates for the primary analysis are drawn are included in Table 4-7.

Premature Mortality. Both long- and short-term exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates from epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make mortality risk reduction the most significant health endpoint quantified in this analysis.

Although a number of uncertainties remain to be addressed by continued research (NRC, 1998), a substantial body of published scientific literature documents the correlation between elevated PM concentrations and increased mortality rates. Time-series methods relate short-term (often day-to-day) changes in PM concentrations and changes in daily mortality rates up to several days after a period of elevated PM concentrations. Cohort methods examine the potential relationship between community-level PM exposures over multiple years (i.e., long-term exposures) and community-level annual mortality rates. Researchers have found statistically significant associations between PM and premature mortality using both types of studies. In general, the risk estimates based on the cohort studies are larger than those derived from time-series studies. Cohort analyses are thought to better capture the full public health impact of exposure to air pollution over time, because they capture the effects of long-term exposures and possibly some component of short-term exposures (Kunzli et al., 2001; NRC, 2002). This section discusses some of the issues surrounding the estimation of premature mortality.

Over a dozen studies have found significant associations between various measures of long-term exposure to PM and elevated rates of annual mortality, beginning with Lave and Seskin (1977). Most of the published studies found positive (but not always statistically

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Premature mortality —cohort study, all- cause	PM _{2.5} (annual mean)	Pope et al. (2002)	>29 years
Premature mortality — all-cause	PM _{2.5} (annual mean)	Woodruff et al. (1997)	Infant (<1 year
Chronic Illness			
Chronic bronchitis	PM _{2.5}	Abbey et al. (1995)	>26 years
Nonfatal heart attacks	PM _{2.5}	Peters et al. (2001)	Adults
Hospital Admissions			
Respiratory	Ozone	Pooled estimate: Schwartz (1995)—ICD 460-519 (all resp) Schwartz (1994a, 1994b)—ICD 480-486 (pneumonia) Moolgavkar et al. (1997)—ICD 480-487 (pneumonia) Schwartz (1994b)—ICD 491-492, 494-496 (COPD) Moolgavkar et al. (1997)—ICD 490-496 (COPD)	>64 years
	Ozone	Burnett et al. (2001)	<2 years
	PM _{2.5}	Pooled estimate: Moolgavkar (2003)—ICD 490-496 (COPD) Ito (2003)—ICD 490-496 (COPD)	>64 years
	PM _{2.5}	Moolgavkar (2000)—ICD 490-496 (COPD)	20-64 years
	PM _{2.5}	Ito (2003)—ICD 480-486 (pneumonia)	>64 years
	PM _{2.5}	Sheppard (2003)—ICD 493 (asthma)	<65 years
Cardiovascular	PM _{2.5}	Pooled estimate: Moolgavkar (2003)—ICD 390-429 (all cardiovascular) Ito (2003)—ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	>64 years
	PM _{2.5}	Moolgavkar (2000)—ICD 390-429 (all cardiovascular)	20-64 years
Asthma-related ER visits	Ozone	Pooled estimate: Weisel et al. (1995), Cody et al. (1992), Stieb et al. (1996)	All ages
	PM _{2.5}	Norris et al. (1999)	0-18 years

Table 4-7. Endpoints and Studies Used to Calculate Total Monetized Health Benefits

Table 4-7. Endpoints and Studies Used to Calculate Total Monetized Health Benefits (continued)

Endpoint	Pollutant	Study	Study Population
Other Health Endpoints			
Acute bronchitis	PM _{2.5}	Dockery et al. (1996)	8-12 years
Upper respiratory symptoms	PM ₁₀	Pope et al. (1991)	Asthmatics, 9–11 years
Lower respiratory symptoms	PM _{2.5}	Schwartz and Neas (2000)	7–14 years
Asthma exacerbations	PM _{2.5}	Pooled estimate: Ostro et al. (2001) (cough, wheeze and shortness of breath) Vedal et al. (1998) (cough)	6–18 years ^a
Work loss days	PM _{2.5}	Ostro (1987)	18–65 years
School absence days	Ozone	Pooled estimate: Gilliland et al. (2001) Chen et al. (2000)	9–10 years 6–11 years
Worker productivity	Ozone	Crocker and Horst (1981)	Outdoor workers, 18–65
MRADs	PM _{2.5} , Ozone	Ostro and Rothschild (1989)	18–65 years

^a The original study populations were 8 to 13 for the Ostro et al. (2001) study and 6 to 13 for the Vedal et al. (1998) study. Based on advice from the SAB-HES, we extended the applied population to 6 to 18, reflecting the common biological basis for the effect in children in the broader age group.

significant) associations with available PM indices such as total suspended particles (TSP). However, exploration of alternative model specifications sometimes raised questions about causal relationships (e.g., Lipfert, Morris, and Wyzga [1989]). These early "cross-sectional" studies (e.g., Lave and Seskin [1977]; Ozkaynak and Thurston [1987]) were criticized for a number of methodological limitations, particularly for inadequate control at the individual level for variables that are potentially important in causing mortality, such as wealth, smoking, and diet. More recently, several studies have been published that use improved approaches and appear to be consistent with the earlier body of literature. These new "prospective cohort" studies reflect a significant improvement over the earlier work because they include individual-level information with respect to health status and residence. The most extensive analyses have been based on data from two prospective cohort groups, often

referred to as the Harvard "Six-Cities Study" (Dockery et al., 1993) and the "American Cancer Society or ACS study" (Pope et al., 1995); these studies have found consistent relationships between fine particle indicators and premature mortality across multiple locations in the United States. A third major data set comes from the California-based 7th Day Adventist Study (e.g., Abbey et al., 1999), which reported associations between long-term PM exposure and mortality in men. Results from this cohort, however, have been inconsistent, and the air quality results are not geographically representative of most of the United States. More recently, a cohort of adult male veterans diagnosed with hypertension has been examined (Lipfert et al., 2000). The characteristics of this group differ from the cohorts in the Six-Cities, ACS, and 7th Day Adventist studies with respect to income, race, health status, and smoking status. Unlike previous long-term analyses, this study found some associations between mortality and ozone but found inconsistent results for PM indicators. Because of the selective nature of the population in the veteran's cohort, we have chosen not to include any effect estimates from the Lipfert et al. (2000) study in our benefits assessment.¹²

Given their consistent results and broad geographic coverage, the Six-Cities and ACS data have been particularly important in benefits analyses. The credibility of these two studies is further enhanced by the fact that they were subject to extensive reexamination and reanalysis by an independent team of scientific experts commissioned by HEI (Krewski et al., 2000). The final results of the reanalysis were then independently peer reviewed by a Special Panel of the HEI Health Review Committee. The results of these reanalyses confirmed and expanded those of the original investigators. This intensive independent reanalysis effort was occasioned both by the importance of the original findings and concerns that the underlying individual health effects information has never been made publicly available.

¹²EPA recognizes that the ACS cohort also is not representative of the demographic mix in the general population. The ACS cohort is almost entirely white and has higher income and education levels relative to the general population. EPA's approach to this problem is to match populations based on the potential for demographic characteristics to modify the effect of air pollution on mortality risk. Thus, for the various ACS-based models, we are careful to apply the effect estimate only to ages matching those in the original studies, because age has a potentially large modifying impact on the effect estimate, especially when younger individuals are excluded from the study population. For the Lipfert analysis, the applied population should be limited to that matching the sample used in the analysis. This sample was all male, veterans, and diagnosed hypertensive. There are also a number of differences between the composition of the sample and the general population, including a higher percentage of African Americans (35 percent) and a much higher percentage of smokers (81 percent former smokers, 57 percent current smokers) than the general population (12 percent African American, 24 percent current smokers).

While the HEI reexamination lends credibility to the original studies, it also highlights sensitivities concerning the relative impact of various pollutants, the potential role of education in mediating the association between pollution and mortality, and the influence of spatial correlation modeling. Further confirmation and extension of the overall findings using more recent air quality and a longer follow-up period for the ACS cohort was recently published (Pope et al., 2002).

In developing and improving the methods for estimating and valuing the potential reductions in mortality risk over the years, EPA consulted with the SAB-HES. That panel recommended using long-term prospective cohort studies in estimating mortality risk reduction (EPA-SAB-COUNCIL-ADV-99-005, 1999). This recommendation has been confirmed by a recent report from the National Research Council, which stated that "it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure" (NRC, 2002, p. 108). More specifically, the SAB recommended emphasis on the ACS study because it includes a much larger sample size and longer exposure interval and covers more locations (e.g., 50 cities compared to the Six-Cities Study) than other studies of its kind. As explained in the regulatory impact analysis for the Heavy-Duty Engine/Diesel Fuel rule (EPA, 2000d), more recent EPA benefits analyses have relied on an improved specification of the ACS cohort data that was developed in the HEI reanalysis (Krewski et al., 2000). The latest reanalysis of the ACS cohort data (Pope et al., 2002) provides additional refinements to the analysis of PM-related mortality by a) extending the follow-up period for the ACS study subjects to 16 years, which triples the size of the mortality data set; b) substantially increasing exposure data, including consideration for cohort exposure to PM25 following implementation of the PM25 standard in 1999; c) controlling for a variety of personal risk factors including occupational exposure and diet; and d) using advanced statistical methods to evaluate specific issues that can adversely affect risk estimates including the possibility of spatial autocorrelation of survival times in communities located near each other. Because of these refinements, the SAB-HES recommends using the Pope et al. (2002) study as the basis for the primary mortality estimate for adults and suggests that alternate estimates of mortality generated using other cohort and time-series studies could be included as part of the sensitivity analysis (SAB-HES, 2004).

The SAB-HES also recommended using the estimated relative risks from the Pope et al. (2002) study based on the average exposure to $PM_{2.5}$, measured by the average of two $PM_{2.5}$ measurements, over the periods 1979–1983 and 1999–2000. In addition to relative risks for all-cause mortality, the Pope et al. (2002) study provides relative risks for cardiopulmonary, lung cancer, and all-other cause mortality. Because of concerns regarding

the statistical reliability of the all-other cause mortality relative risk estimates, we calculated mortality impacts for the primary analysis based on the all-cause relative risk. However, we provide separate estimates of cardiopulmonary and lung cancer deaths to show how these important causes of death are affected by reductions in $PM_{2.5}$.

Recently published studies have strengthened the case for an association between PM exposure and respiratory inflamation and infection leading to premature mortality in children under 5 years of age. Specifically, the SAB-HES noted the release of the WHO Global Burden of Disease Study focusing on ambient air, which cites several recently published time-series studies relating daily PM exposure to mortality in children (SAB-HES, 2003). The SAB-HES also cites the study by Belanger et al. (2003) as corroborating findings linking PM exposure to increased respiratory inflamation and infections in children. Recently, a study by Chay and Greenstone (2003) found that reductions in TSP caused by the recession of 1981–1982 were related to reductions in infant mortality at the county level. With regard to the cohort study conducted by Woodruff et al. (1997), the SAB-HES notes several strengths of the study, including the use of a larger cohort drawn from a large number of metropolitan areas and efforts to control for a variety of individual risk factors in infants (e.g., maternal educational level, maternal ethnicity, parental marital status, and maternal smoking status). Based on these findings, the SAB-HES recommends that EPA incorporate infant mortality into the primary benefits estimate and that infant mortality be evaluated using an impact function developed from the Woodruff et al. (1997) study (SAB-HES, 2004).

Chronic Bronchitis. CB is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 5 percent of the U.S. population (American Lung Association, 1999). A limited number of studies have estimated the impact of air pollution on new incidences of CB. Schwartz (1993) and Abbey et al. (1995) provide evidence that long-term PM exposure gives rise to the development of CB in the United States. Because the CAIR is expected to reduce primarily $PM_{2.5}$, this analysis uses only the Abbey et al. (1995) study, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of CB.

Nonfatal Myocardial Infarctions (heart attacks). Nonfatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States (Peters et al., 2001) and other countries (Poloniecki et al., 1997). We used a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between $PM_{2.5}$ and nonfatal heart attacks. Peters et al. is the only available U.S. study to provide a specific estimate for heart

attacks. Other studies, such as Samet et al. (2000) and Moolgavkar (2000), show a consistent relationship between all cardiovascular hospital admissions, including those for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on long-term health costs and earnings, we provide a separate estimate for nonfatal heart attacks. The estimate used in the CAIR analysis is based on the single available U.S. effect estimate. The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the United States. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other coronary heart diseases (Carthenon et al., 2002; Dekker et al., 2000; Liao et al., 1997; Tsuji et al., 1996). As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

Hospital and Emergency Room Admissions. Because of the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, many of the hospital admission endpoints use pooled impact functions based on the results of a number of studies. In addition, some studies have examined the relationship between air pollution and emergency room visits. Since most emergency room visits do not result in an admission to the hospital (the majority of people going to the emergency room are treated and return home), we treat hospital admissions and emergency room visits separately, taking account of the fraction of emergency room visits that are admitted to the hospital.

The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking ozone or PM with other types of hospital admissions. The only type of emergency room visits that have been consistently linked to ozone and PM in the United States are asthma-related visits.

To estimate avoided incidences of cardiovascular hospital admissions associated with $PM_{2.5}$, we used studies by Moolgavkar (2003) and Ito (2003). Additional published studies show a statistically significant relationship between PM_{10} and cardiovascular hospital admissions. However, given that the control options we are analyzing are expected to reduce primarily $PM_{2.5}$, we focus on the two studies that examine $PM_{2.5}$. Both of these studies provide an effect estimate for populations over 65, allowing us to pool the impact functions for this age group. Only Moolgavkar (2000) provided a separate effect estimate for

populations 20 to 64.¹³ Total cardiovascular hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Cardiovascular hospital admissions include admissions for myocardial infarctions. To avoid double-counting benefits from reductions in myocardial infarctions when applying the impact function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for myocardial infarctions.

To estimate total avoided incidences of respiratory hospital admissions, we used impact functions for several respiratory causes, including chronic obstructive pulmonary disease (COPD), pneumonia, and asthma. As with cardiovascular admissions, additional published studies show a statistically significant relationship between PM_{10} and respiratory hospital admissions. We used only those focusing on $PM_{2.5}$. Both Moolgavkar (2000) and Ito (2003) provide effect estimates for COPD in populations over 65, allowing us to pool the impact functions for this group. Only Moolgavkar (2000) provides a separate effect estimate for populations 20 to 64. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Only Ito (2003) estimated pneumonia and only for the population 65 and older. In addition, Sheppard (2003) provided an effect estimate for asthma hospital admissions for populations under age 65. Total avoided incidences of PM-related respiratory-related hospital admissions is the sum of COPD, pneumonia, and asthma admissions.

To estimate the effects of PM air pollution reductions on asthma-related ER visits, we use the effect estimate from a study of children 18 and under by Norris et al. (1999). As noted earlier, there is another study by Schwartz examining a broader age group (less than 65), but the Schwartz study focused on PM_{10} rather than $PM_{2.5}$. We selected the Norris et al. (1999) effect estimate because it better matched the pollutant of interest. Because children tend to have higher rates of hospitalization for asthma relative to adults under 65, we will likely capture the majority of the impact of $PM_{2.5}$ on asthma emergency room visits in populations under 65, although there may still be significant impacts in the adult population under 65.

¹³Note that the Moolgavkar (2000) study has not been updated to reflect the more stringent GAM convergence criteria. However, given that no other estimates are available for this age group, we chose to use the existing study. Given the very small (<5 percent) difference in the effect estimates for people 65 and older with cardiovascular hospital admissions between the original and reanalyzed results, we do not expect this choice to introduce much bias.</p>

To estimate avoided incidences of respiratory hospital admissions associated with ozone, we used a number of studies examining hospital admissions for a range of respiratory illnesses, including pneumonia and COPD. Two age groups, adults over 65 and children under 2, were examined. For adults over 65, Schwartz (1995) provides effect estimates for two different cities relating ozone and hospital admissions for all respiratory causes (defined as ICD codes 460-519). Impact functions based on these studies were pooled first before being pooled with other studies. Two studies (Moolgavkar et al., 1997; Schwartz, 1994a) examine ozone and pneumonia hospital admissions in Minneapolis. One additional study (Schwartz, 1994b) examines ozone and pneumonia hospital admissions in Detroit. The impact functions for Minneapolis were pooled together first, and the resulting impact function was then pooled with the impact function for Detroit. This avoids assigning too much weight to the information coming from one city. For COPD hospital admissions, two studies are available: Moolgavkar et al. (1997), conducted in Minneapolis, and Schwartz (1994b), conducted in Detroit. These two studies were pooled together. To estimate total respiratory hospital admissions for adults over 65, COPD admissions were added to pneumonia admissions, and the result was pooled with the Schwartz (1995) estimate of total respiratory admissions. Burnett et al. (2001) is the only study providing an effect estimate for respiratory hospital admissions in children under 2.

Acute Health Events and School/Work Loss Days. As indicated in Table 4-1, in addition to mortality, chronic illness, and hospital admissions, a number of acute health effects not requiring hospitalization are associated with exposure to ambient levels of ozone and PM. The sources for the effect estimates used to quantify these effects are described below.

Around 4 percent of U.S. children between the ages of 5 and 17 experience episodes of acute bronchitis annually (American Lung Association, 2002c). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia,¹⁴ with the exception of cough, most acute bronchitis symptoms abate within 7 to 10 days. Incidence of episodes of acute bronchitis in children between the ages of 5 and 17 were estimated using an effect estimate developed from Dockery et al. (1996).

Incidences of lower respiratory symptoms (e.g., wheezing, deep cough) in children aged 7 to 14 were estimated using an effect estimate from Schwartz and Neas (2000).

¹⁴See http://www.nlm.nih.gov/medlineplus/ency/article/000124.htm, accessed January 2002.

Because asthmatics have greater sensitivity to stimuli (including air pollution), children with asthma can be more susceptible to a variety of upper respiratory symptoms (e.g., runny or stuffy nose; wet cough; and burning, aching, or red eyes). Research on the effects of air pollution on upper respiratory symptoms has thus focused on effects in asthmatics. Incidences of upper respiratory symptoms in asthmatic children aged 9 to 11 are estimated using an effect estimate developed from Pope et al. (1991).

Health effects from air pollution can also result in missed days of work (either from personal symptoms or from caring for a sick family member). Days of work lost due to $PM_{2.5}$ were estimated using an effect estimate developed from Ostro (1987). Children may also be absent from school because of respiratory or other diseases caused by exposure to air pollution. Most studies examining school absence rates have found little or no association with $PM_{2.5}$, but several studies have found a significant association between ozone levels and school absence rates. We used two recent studies, Gilliland et al. (2001) and Chen et al. (2000), to estimate changes in absences (school loss days) due to changes in ozone levels. The Gilliland et al. study estimated the incidence of new periods of absence, while the Chen et al. study examined absence periods by the average duration of an absence. We estimated an average duration of school absence of 1.6 days by dividing the average daily school absence rate from Chen et al. (2001). This provides estimates from Chen et al. (2000) and Gilliland et al. (2001), which can be pooled to provide an overall estimate.

MRAD result when individuals reduce most usual daily activities and replace them with less strenuous activities or rest, yet not to the point of missing work or school. For example, a mechanic who would usually be doing physical work most of the day will instead spend the day at a desk doing paper and phone work because of difficulty breathing or chest pain. The effect of PM_{2.5} and ozone on MRAD was estimated using an effect estimate derived from Ostro and Rothschild (1989).

For CAIR, we have followed the SAB-HES recommendations regarding asthma exacerbations in developing the primary estimate. To prevent double-counting, we focused the estimation on asthma exacerbations occurring in children and excluded adults from the calculation.¹⁵ Asthma exacerbations occurring in adults are assumed to be captured in the

¹⁵Estimating asthma exacerbations associated with air pollution exposures is difficult, due to concerns about double-counting of benefits. Concerns over double-counting stem from the fact that studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly

general population endpoints such as work loss days and MRADs. Consequently, if we had included an adult-specific asthma exacerbation estimate, we would likely double-count incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double-counting.

To characterize asthma exacerbations in children, we selected two studies (Ostro et al., 2001; Vedal et al., 1998) that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between PM₂₅, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this endpoint, along with shortness of breath and wheeze, in generating incidence estimates (see below). Vedal et al. (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Association between PM₁₀ and respiratory symptoms for the asthmatic population was only reported for two endpoints: cough and PEF. Because it is difficult to translate PEF measures into clearly defined health endpoints that can be monetized, we only included the cough-related effect estimate from this study in quantifying asthma exacerbations. We

added to the general population numbers without double-counting. In one specific case (upper respiratory symptoms in children), the only study available is limited to asthmatic children, so this endpoint can be readily included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms and MRADs, are estimated for the total population that includes asthmatics. Therefore, to simply add predictions of asthma-related symptoms generated for the population of asthmatics to these total population-based estimates could result in double-counting, especially if they evaluate similar endpoints. The SAB-HES, in commenting on the analytical blueprint for 812, acknowledged these challenges in evaluating asthmatic symptoms and appropriately adding them into the primary analysis (SAB-HES, 2004). However, despite these challenges, the SAB-HES recommends the addition of asthma-related symptoms (i.e., asthma exacerbations) to the primary analysis, provided that the studies use the panel study approach and that they have comparable design and baseline frequencies in both asthma prevalence and exacerbation rates. Note also, that the SAB-HES, while supporting the incorporation of asthma exacerbation estimates, does not believe that the association between ambient air pollution, including ozone and PM, and the new onset of asthma is sufficiently strong to support inclusion of this asthma-related endpoint in the primary estimate.

employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro et al. study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro et al. study is then pooled with the cough-related estimate generated using the Vedal study. The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

Additional epidemiological studies are available for characterizing asthma-related health endpoints (the full list of epidemiological studies considered for modeling asthmarelated incidence is presented in Table 4-8). However, based on recommendations from the SAB-HES, we decided not to use these additional studies in generating the primary estimate. In particular, the Yu et al. (2000) estimates show a much higher baseline incidence rate than other studies, which may lead to an overstatement of the expected impacts in the overall asthmatic population. The Whittemore and Korn (1980) study did not use a well-defined endpoint, instead focusing on a respondent-defined "asthma attack." Other studies looked at respiratory symptoms in asthmatics but did not focus on specific exacerbations of asthma.

4.1.5.2 Uncertainties Associated with Health Impact Functions

Within-Study Variation. Within-study variation refers to the precision with which a given study estimates the relationship between air quality changes and health effects. Health effects studies provide both a "best estimate" of this relationship plus a measure of the statistical uncertainty of the relationship. The size of this uncertainty depends on factors such as the number of subjects studied and the size of the effect being measured. The results of even the most well-designed epidemiological studies are characterized by this type of uncertainty, though well-designed studies typically report narrower uncertainty bounds around the best estimate than do studies of lesser quality. In selecting health endpoints, we generally focus on endpoints where a statistically significant relationship has been observed in at least some studies, although we may pool together results from studies with both statistically significant and insignificant estimates to avoid selection bias.

Across-Study Variation. Across-study variation refers to the fact that different published studies of the same pollutant/health effect relationship typically do not report identical findings; in some instances the differences are substantial. These differences can exist even between equally reputable studies and may result in health effect estimates that

Endpoint	Definition	Pollutant	Study	Study Population		
Asthma Attack Indicators						
Shortness of breath	Prevalence of shortness of breath; incidence of shortness of breath	PM _{2.5}	Ostro et al. (2001)	African-American asthmatics, 8–13		
Cough	Prevalence of cough; incidence of cough	PM _{2.5}	Ostro et al. (2001)	African-American asthmatics, 8–13		
Wheeze	Prevalence of wheeze; incidence of wheeze	PM _{2.5}	Ostro et al. (2001)	African-American asthmatics, 8–13		
Asthma exacerbation	≥1 mild asthma symptom: wheeze, cough, chest tightness, shortness of breath	PM ₁₀ , PM _{1.0}	Yu et al. (2000)	Asthmatics, 5–13		
Cough	Prevalence of cough	PM_{10}	Vedal et al. (1998)	Asthmatics, 6–13		
Other Symptoms/Illn	ness Endpoints					
Upper respiratory symptoms	≥1 of the following: runny or stuffy nose; wet cough; burning, aching, or red eyes	PM ₁₀	Pope et al. (1991)	Asthmatics, 9–11		
Moderate or worse asthma	Probability of moderate (or worse) rating of overall asthma status	PM _{2.5}	Ostro et al. (1991)	Asthmatics, all ages		
Acute bronchitis	\geq 1 episodes of bronchitis in the past 12 months	PM _{2.5}	McConnell et al. (1999)	Asthmatics, 9–15		
Phlegm	"Other than with colds, does this child usually seem congested in the chest or bring up phlegm?"	PM _{2.5}	McConnell et al. (1999)	Asthmatics, 9–15		
Asthma attacks	Respondent-defined asthma attack	PM _{2.5} , ozone	Whittemore and Korn (1980)	Asthmatics, all ages		

 Table 4-8. Studies Examining Health Impacts in the Asthmatic Population Evaluated

 for Use in the Benefits Analysis

vary considerably. Across-study variation can result from two possible causes. One possibility is that studies report different estimates of the single true relationship between a given pollutant and a health effect because of differences in study design, random chance, or other factors. For example, a hypothetical study conducted in New York and one conducted in Seattle may report different C-R functions for the relationship between PM and mortality, in part because of differences between these two study populations (e.g., demographics, activity patterns). Alternatively, study results may differ because these two studies are in fact estimating different relationships; that is, the same reduction in PM in New York and

Seattle may result in different reductions in premature mortality. This may result from a number of factors, such as differences in the relative sensitivity of these two populations to PM pollution and differences in the composition of PM in these two locations. In either case, where we identified multiple studies that are appropriate for estimating a given health effect, we generated a pooled estimate of results from each of those studies.

Application of C-R Relationship Nationwide. Regardless of the use of impact functions based on effect estimates from a single epidemiological study or multiple studies, each impact function was applied uniformly throughout the United States to generate health benefit estimates. However, to the extent that pollutant/health effect relationships are region specific, applying a location-specific impact function at all locations in the United States may result in overestimates of health effect changes in some locations and underestimates of health effect changes in other locations. It is not possible, however, to know the extent or direction of the overall effect on health benefit estimates introduced by applying a single impact function to the entire United States. This may be a significant uncertainty in the analysis, but the current state of the scientific literature does not allow for a region-specific estimation of health benefits.¹⁶

Extrapolation of Impact Functions Across Populations. Epidemiological studies often focus on specific age ranges, either due to data availability limitations (e.g., most hospital admission data come from Medicare records, which are limited to populations 65 and older), or to simplify data collection (e.g., some asthma symptom studies focus on children at summer camps, which usually have a limited age range). We have assumed for the primary analysis that most impact functions should be applied only to those populations with ages that strictly match the populations in the underlying epidemiological studies. However, in many cases, there is no biological reason why the observed health effect would not also occur in other populations within a reasonable range of the studied population. For example, Dockery et al. (1996) examined acute bronchitis in children aged 8 to 12. There is no biological reason to expect a very different response in children aged 6 or 14. By excluding populations outside the range in the studies, we may be underestimating the health impact in the overall population. In response to recommendations from the SAB-HES, where there appears to be a reasonable physiological basis for expanding the age group associated with a specific effect estimate beyond the study population to cover the full age

¹⁶Although we are not able to use region-specific effect estimates, we use region-specific baseline incidence rates where available. This allows us to take into account regional differences in health status, which can have a significant impact on estimated health benefits.

group (e.g., expanding from a study population of 7 to 11 year olds to the full 6- to 18-year child age group), we have done so and used those expanded incidence estimates in the primary analysis.

Uncertainties in the PM Mortality Relationship. A substantial body of published scientific literature demonstrates a correlation between elevated PM concentrations and increased premature mortality. However, much about this relationship is still uncertain. These uncertainties include the following:

<u>Causality</u>: Epidemiological studies are not designed to definitively prove causation. For the analysis of the CAIR, we assumed a causal relationship between exposure to elevated PM and premature mortality, based on the consistent evidence of a correlation between PM and mortality reported in the substantial body of published scientific literature.

<u>Other Pollutants</u>: PM concentrations are correlated with the concentrations of other criteria pollutants, such as ozone and CO. To the extent that there is correlation, this analysis may be assigning mortality effects to PM exposure that are actually the result of exposure to other pollutants. Recent studies (see Thurston and Ito [2001] and Bell et al [2004]) have explored whether ozone may have mortality effects independent of PM. EPA is currently evaluating the epidemiological literature on the relationship between ozone and mortality.

Shape of the C-R Function: The shape of the true PM mortality C-R function is uncertain, but this analysis assumes the C-R function has a non-threshold log-linear form throughout the relevant range of exposures. If this is not the correct form of the C-R function, or if certain scenarios predict concentrations well above the range of values for which the C-R function was fitted, avoided mortality may be misestimated. Although not included in the primary analysis, the potential impact of a health effects threshold on avoided incidences of PM-related premature mortality is explored as a key sensitivity analysis.

The possible existence of an effect threshold is a very important scientific question and issue for policy analyses such as this one. In 1999, the EPA SAB Advisory Council for Clean Air Compliance advised EPA that there was currently no scientific basis for selecting a threshold of 15 μ g/m³ or any other specific threshold for the PM-related health effects considered in typical benefits analyses (EPA-SAB-Council-ADV-99-012, 1999). In 2000, as a part of their review of benefits methods, the National Research Council concluded that there is no evidence for any departure from linearity in the observed range of exposure to PM₁₀ or PM_{2.5}, nor any indication of a threshold (NRC, 2002). They cite the weight of evidence available from both short- and long-term exposure models and the similar effects found in cities with low and high ambient concentrations of PM. Most recently, EPA's updated (2004) Criteria Document states, "In summary, the available evidence does not either support or refute the existence of thresholds for effects of PM on mortality across the range of uncertainties in the studies." The PM criteria document identifies the general shape of exposure-response relationship(s) between PM and/or other pollutants and observed health effects (e.g., potential indications of thresholds), as an important issue and uncertainty in interpreting the overall PM epidemiology database.

These recommendations are supported by the recent literature on health effects of short and longer term PM exposures (Daniels et al., 2000; Pope, 2000; Pope et al, 2002; Rossi et al., 1999; Schwartz and Zanobetti, 2000; Schwartz, Laden, and Zanobetti, 2002; Smith et al. 2000) that finds in most cases no evidence of a nonlinear relationship between PM and health effects and certainly does not find a distinct threshold. Recent cohort analyses by HEI (Krewski et al., 2000) and Pope et al. (2002) provide additional evidence of a quasi-linear relationship between long-term exposures to PM_{25} and mortality. According to the latest draft PM criteria document, Krewski et al. (2000) found a "found a visually near-linear relationship between all-cause and cardiopulmonary mortality residuals and mean sulfate concentrations, near-linear between cardiopulmonary mortality and mean PM2.5, but a somewhat nonlinear relationship between all-cause mortality residuals and mean PM2.5 concentrations that flattens above $\sim 20 \ \mu g/m3$. The confidence bands around the fitted curves are very wide, however, neither requiring a linear relationship nor precluding a nonlinear relationship if suggested by reanalyses" (Krewski et al. (2000), page 8-138). The Pope et al. (2002) analysis, which represented an extension to the Krewski et al. analysis, found that the functions relating PM_{2.5} and mortality are not significantly different from linear associations.

Based on the recent literature and advice from the SAB, we assume there are no thresholds for modeling health effects. Although not included in the primary analysis, the potential impact of a health effects threshold on avoided incidences of PM-related premature mortality is explored as a key sensitivity analysis.

<u>Regional Differences</u>: As discussed above, significant variability exists in the results of different PM/mortality studies. This variability may reflect regionally specific C-R functions resulting from regional differences in factors such as the physical and chemical composition of PM. If true regional differences exist, applying the PM/mortality C-R function to regions outside the study location could result in misestimation of effects in these regions. **US EPA ARCHIVE DOCUMENT**

Exposure/Mortality Lags: There is a time lag between changes in PM exposures and the total realization of changes in annual mortality rates. For the chronic PM/mortality relationship, the length of the lag is unknown and may be dependent on the kind of exposure. The existence of such a lag is important for the valuation of premature mortality incidence because economic theory suggests that benefits occurring in the future should be discounted. There is no specific scientific evidence of the existence or structure of a PM effects lag. However, current scientific literature on adverse health effects similar to those associated with PM (e.g., smoking-related disease) and the difference in the effect size between chronic exposure studies and daily mortality studies suggests that all incidences of premature mortality reduction associated with a given incremental change in PM exposure probably would not occur in the same year as the exposure reduction. The smoking-related literature also implies that lags of up to a few years or longer are plausible. The SAB-HES suggests that appropriate lag structures may be developed based on the distribution of cause-specific deaths within the overall all-cause estimate. Diseases with longer progressions should be characterized by long-term lag structures, while impacts occurring in populations with existing disease may be characterized by short-term lags.

A key question is the distribution of causes of death within the relatively broad categories analyzed in the cohort studies used. While we may be more certain about the appropriate length of cessation lag for lung cancer deaths, it is not clear what the appropriate lag structure should be for different types of cardiopulmonary deaths, which include both respiratory and cardiovascular causes. Some respiratory diseases may have a long period of progression, while others, such as pneumonia, have a very short duration. In the case of cardiovascular disease, there is an important question of whether air pollution is causing the disease, which would imply a relatively long cessation lag, or whether air pollution is causing premature death in individuals with preexisting heart disease, which would imply very short cessation lags.

The SAB-HES provides several recommendations for future research that could support the development of defensible lag structures, including the use of disease-specific lag models, and the construction of a segmented lag distribution to combine differential lags across causes of death. The SAB-HES recommended that until additional research has been completed, EPA should assume a segmented lag structure characterized by 30 percent of mortality reductions occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in $PM_{2.5}$, and 20 percent occurring evenly over the years 6 to 20 after the reduction in $PM_{2.5}$. The distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to

5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. For future analyses, the specific distribution of deaths over time will need to be determined through research on causes of death and progression of diseases associated with air pollution. It is important to keep in mind that changes in the lag assumptions do not change the total number of estimated deaths but rather the timing of those deaths.

<u>Cumulative Effects</u>: We attribute the PM/mortality relationship in the underlying epidemiological studies to cumulative exposure to PM. However, the relative roles of PM exposure duration and PM exposure level in inducing premature mortality remain unknown at this time.

4.1.5.3 Baseline Health Effect Incidence Rates

The epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the relative risk of a health effect, rather than an estimate of the absolute number of avoided cases. For example, a typical result might be that a 10 μ g/m³ decrease in daily PM_{2.5} levels might decrease hospital admissions by 3 percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. The baseline incidence rate provides an estimate of the incidence rate (number of cases of the health effect per year, usually per 10,000 or 100,000 general population) in the assessment location corresponding to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number (e.g., if the baseline incidence rate is number of cases per year per 100,000 population, it must be multiplied by the number of 100,000 sin the population).

Some epidemiological studies examine the association between pollution levels and adverse health effects in a specific subpopulation, such as asthmatics or diabetics. In these cases, it is necessary to develop not only baseline incidence rates, but also prevalence rates for the defining condition (e.g., asthma). For both baseline incidence and prevalence data, we use age-specific rates where available. Impact functions are applied to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits.

In most cases, because of a lack of data or methods, we have not attempted to project incidence rates to future years, instead assuming that the most recent data on incidence rates is the best prediction of future incidence rates. In recent years, better data on trends in incidence and prevalence rates for some endpoints, such as asthma, have become available.

We are working to develop methods to use these data to project future incidence rates. However, for our primary benefits analysis of the final CAIR, we continue to use current incidence rates.

Table 4-9 summarizes the baseline incidence data and sources used in the benefits analysis. We use the most geographically disaggregated data available. For premature mortality, county-level data are available. For hospital admissions, regional rates are available. However, for all other endpoints, a single national incidence rate is used, due to a lack of more spatially disaggregated data. In these cases, we used national incidence rates whenever possible, because these data are most applicable to a national assessment of benefits. However, for some studies, the only available incidence information comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level.

Age, cause, and county-specific mortality rates were obtained from the U.S. Centers for Disease Control and Prevention (CDC) for the years 1996 through 1998. CDC maintains an online data repository of health statistics, CDC Wonder, accessible at http://wonder.cdc.gov/. The mortality rates provided are derived from U.S. death records and U.S. Census Bureau postcensal population estimates. Mortality rates were averaged across 3 years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group. For example, to estimate mortality rates for individuals ages 30 and up, we scaled the 25- to 34-year-old death count and population by one-half and then generated a population-weighted mortality rate using data for the older age groups. Note that we have not projected any changes in mortality rates over time. We are aware that the U.S. Census projections of total and age-specific mortality rates used in our population projections are based on projections of declines in mortality rates for younger populations and increases in mortality rates for older populations over time. We are evaluating the most appropriate way to incorporate these projections into our database of county-level cause-specific mortality rates. In the interim, we have not attempted to adjust future mortality rates. This will lead to an overestimate of mortality benefits in future years, with the overestimation bias increasing as benefits are projected into the future. We do not at this time have a quantified estimate of the magnitude of the potential bias in the years analyzed for this rule (2010 and 2015).

		Rates			
Endpoint	Parameter	Value	Source ^a		
Mortality	Daily or annual mortality rate	Age-, cause-, and county-specific rate	CDC Wonder (1996–1998)		
Hospitalizations	Daily hospitalization rate	Age-, region-, and cause-specific rate	1999 NHDS public use data files ^b		
Asthma ER Visits	Daily asthma ER visit rate	Age- and region- specific visit rate	2000 NHAMCS public use data files ^c ; 1999 NHDS public use data files ^b		
Chronic Bronchitis	 Annual prevalence rate per person Aged 18–44 Aged 45–64 Aged 65 and older 	0.0367 0.0505 0.0587	1999 NHIS (American Lung Association, 2002b, Table 4)		
	Annual incidence rate per person	0.00378	Abbey et al. (1993b, Table 3)		
Nonfatal Myocardial Infarction (heart attacks)	Daily nonfatal myocardial infarction incidence rate per person, 18+ • Northeast • Midwest • South • West	0.0000159 0.0000135 0.0000111 0.0000100	1999 NHDS public use data files ^b ; adjusted by 0.93 for probability of surviving after 28 days (Rosamond et al., 1999)		
Asthma Exacerbations	Incidence (and prevalence) among asthmatic African- American children • daily wheeze • daily cough • daily dyspnea	0.076 (0.173) 0.067 (0.145) 0.037 (0.074)	Ostro et al. (2001)		
	Prevalence among asthmatic childrendaily wheezedaily coughdaily dyspnea	0.038 0.086 0.045	Vedal et al. (1998)		
Acute Bronchitis	Annual bronchitis incidence rate, children	0.043	American Lung Association (2002c, Table 11) (continued		

Table 4-9. Baseline Incidence Rates and Population Prevalence Rates for Use in Impact **Functions, General Population**

Rates Endpoint Parameter Value Source^a 0.0012 Lower Daily lower respiratory Schwartz et al. (1994, Table Respiratory symptom incidence 2) Symptoms among children^d Daily upper respiratory 0.3419 Pope et al. (1991, Table 2) Upper Respiratory symptom incidence Symptoms among asthmatic children Work Loss Days 1996 HIS (Adams et al., 1999, Daily WLD incidence Table 41); U.S. Bureau of the rate per person (18-65)0.00540 Census (2000) Aged 18-24 Aged 25-44 0.00678 Aged 45-64 0.00492 Minor Daily MRAD incidence 0.02137 Ostro and Rothschild (1989, Restrictedrate per person p. 243) Activity Days School Loss Daily school absence rate 0.055 National Center for Education Days^e per person Statistics (1996) Daily illness-related 1996 HIS (Adams et al., 1999, school absence rate per Table 47); estimate of 180 person^e 0.0136 school days per year Northeast 0.0146 Midwest 0.0142 South 0.0206 Southwest Daily respiratory illness-1996 HIS (Adams et al., 1999, Table 47); estimate of 180 related school absence rate per person school days per year 0.0073 Northeast 0.0092 Midwest South 0.0061 West 0.0124

 Table 4-9. Baseline Incidence Rates and Population Prevalence Rates for Use in Impact

 Functions, General Population (continued)

^a The following abbreviations are used to describe the national surveys conducted by the National Center for Health Statistics: HIS refers to the National Health Interview Survey; NHDS—National Hospital Discharge Survey; NHAMCS—National Hospital Ambulatory Medical Care Survey.

^b See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/.

° See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/.

^d Lower respiratory symptoms are defined as two or more of the following: cough, chest pain, phlegm, and wheeze.

^e The estimate of daily illness-related school absences excludes school loss days associated with injuries to match the definition in the Gilliland et al. (2001) study.

For the set of endpoints affecting the asthmatic population, in addition to baseline incidence rates, prevalence rates of asthma in the population are needed to define the applicable population. Table 4-9 lists the baseline incidence rates and their sources for asthma symptom endpoints. Table 4-10 lists the prevalence rates used to determine the applicable population for asthma symptom endpoints. Note that these reflect current asthma prevalence and assume no change in prevalence rates in future years. As noted above, we are investigating methods for projecting asthma prevalence rates in future years.

	Asthma Prevalence Rates		
Population Group	Value	Source	
All Ages	0.0386	American Lung Association (2002a, Table 7)—based on 1999 HIS	
< 18	0.0527	American Lung Association (2002a, Table 7)-based on 1999 HIS	
5–17	0.0567	American Lung Association (2002a, Table 7)—based on 1999 HIS	
18–44	0.0371	American Lung Association (2002a, Table 7)—based on 1999 HIS	
45–64	0.0333	American Lung Association (2002a, Table 7)—based on 1999 HIS	
65+	0.0221	American Lung Association (2002a, Table 7)—based on 1999 HIS	
Male, 27+	0.021	2000 HIS public use data files ^a	
African American, 5 to 17	0.0726	American Lung Association (2002a, Table 9)—based on 1999 HIS	
African American, <18	0.0735	American Lung Association (2002a, Table 9)—based on 1999 HIS	

Table 4-10. Asthma Prevalence Rates Used to Estimate Asthmatic Populations inImpact Functions

^a See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHIS/2000/.

4.1.5.4 Selecting Unit Values for Monetizing Health Endpoints

The appropriate economic value for a change in a health effect depends on whether the health effect is viewed *ex ante* (before the effect has occurred) or *ex post* (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a small amount for a large population. The appropriate economic measure is therefore *ex ante* WTP for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk. For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is \$100, then the WTP for an avoided statistical premature mortality amounts to \$1 million (\$100/0.0001 change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. For example, for the valuation of hospital admissions we use the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These COI estimates generally understate the true value of reductions in risk of a health effect. They tend to reflect the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect. Table 4-11 summarizes the value estimates per health effect that we used in this analysis. Values are presented both for a 1990 base income level and adjusted for income growth in the two future analysis years, 2010 and 2015. Note that the unit values for hospital admissions are the weighted averages of the ICD-9 codespecific values for the group of ICD-9 codes included in the hospital admission categories. A discussion of the valuation methods for premature mortality and CB is provided here because of the relative importance of these effects. Discussions of the methods used to value nonfatal myocardial infarctions (heart attacks) and school absence days are provided because these endpoints have only recently been added to the analysis and the valuation methods are still under development. In the following discussions, unit values are presented at 1990 levels of income for consistency with previous analyses. Equivalent future-year values can be obtained from Table 4-11. COI estimates are converted to constant 1999 dollar equivalents using the medical CPI.

4.1.5.4.1 Valuing Reductions in Premature Mortality Risk. We estimate the monetary benefit of reducing premature mortality risk using the VSL approach, which is a summary measure for the value of small changes in mortality risk experienced by a large number of people. The mean value of avoiding one statistical death is assumed to be \$5.5 million in 1999 dollars. This represents a central value consistent with the range of values suggested by recent meta-analyses of the wage-risk VSL literature. The distribution of VSL is characterized by a confidence interval from \$1 to \$10 million, based on two meta-analyses of the wage-risk VSL literature. The limit represents the lower

	Central Estimate of Value Per Statistical Incidence				
Health Endpoint	1990 Income Level	2010 Income Level	2015 Income Level	- Derivation of Estimates	
Premature Mortality (Value of a Statistical Life)	\$5,500,000	\$6,000,000	\$6,400,000	Point estimate is the mean of a normal distribution with a 95 percent confidence interval between \$1 and \$10 million. Confidence interval is based on two meta-analyses of the wage-risk VSL literature: \$1 million represents the lower end of the interquartile range from the Mrozek and Taylor (2002) meta-analysis and \$10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. The VSL represents the value of a small change in mortality risk aggregated over the affected population.	
Chronic Bronchitis (CB)	\$340,000	\$380,000	\$400,000	Point estimate is the mean of a generated distribution of WTP to avoid a case of pollution-related CB. WTP to avoid a case of pollution-related CB is derived by adjusting WTP (as described in Viscusi et al., [1991]) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB.	
Nonfatal Myocardial Infarction (heart attack)				Age-specific cost-of-illness values reflect lost earnings and direct medical costs over a 5-year period following a nonfatal MI. Lost earnings estimates	
<u>3% discount rate</u>	¢((00 0	¢((000	¢((00 0	are based on Cropper and Krupnick (1990). Direct medical costs are based	
Age 0–24	\$66,902	\$66,902	\$66,902	on simple average of estimates from Russell et al. (1998) and Wittels et al.	
Age 25–44	\$74,676	\$74,676	\$74,676	(1990).	
Age 45–54 Age 55–65	\$78,834 \$140,649	\$78,834 \$140,649	\$78,834 \$140,649	<u>Lost earnings</u> : Cropper and Krupnick (1990). Present discounted value of 5 years of lost	
Age 66 and over	\$66,902	\$66,902	\$66,902	earnings:	
Age of and over	\$00,902	\$00,702	\$00,902	age of onset: at 3% at 7%	
7% discount rate				25-44 \$8,774 \$7,855	
Age 0–24	\$65,293	\$65,293	\$65,293	45-54 \$12,932 \$11,578	
Age 25–44	\$73,149	\$73,149	\$73,149	55-65 \$74,746 \$66,920	
Age 45–54	\$76,871	\$76,871	\$76,871	Direct medical expenses: An average of:	
Age 55–65	\$132,214	\$132,214	\$132,214	1. Wittels et al. (1990) (\$102,658—no discounting)	
Age 66 and over	\$65,293	\$65,293	\$65,293	 Russell et al. (1998), 5-year period (\$22,331 at 3% discount rate; \$21,113 at 7% discount rate) 	

Table 4-11. Unit Values Used for Economic Valuation of Health Endpoints (1999\$)

(continued)

	Central Estimate of Value Per Statistical Incidence			_		
Health Endpoint	1990 Income Level	2010 Income Level	2015 Income Level	Derivation of Estimates		
Hospital Admissions						
Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496)	\$12,378	\$12,378	\$12,378	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).		
Pneumonia (ICD codes 480-487)	\$14,693	\$14,693	\$14,693	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total pneumonia category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).		
Asthma Admissions	\$6,634	\$6,634	\$6,634	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).		
All Cardiovascular (ICD codes 390-429)	\$18,387	\$18,387	\$18,387	The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).		
Emergency Room Visits for Asthma	\$286	\$286	\$286	Simple average of two unit COI values: (1) \$311.55, from Smith et al. (1997) and (2) \$260.67, from Stanford et al. (1999).		

Table 4-11. Unit Values Used for Economic Valuation of Health Endpoints (1999\$) (continued)

(continued)

	Central Estimate of Value Per Statistical Incidence			_
Health Endpoint	1990 Income Level	2010 Income Level	2015 Income Level	Derivation of Estimates
Respiratory Ailments Not Requiring	Hospitalization			
Upper Respiratory Symptoms (URS)	\$25	\$26	\$26	Combinations of the three symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in seven different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for URS is the average of the dollar values for the seven different types of URS.
Lower Respiratory Symptoms (LRS)	\$16	\$17	\$17	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS.
Asthma Exacerbations	\$42	\$43	\$44	Asthma exacerbations are valued at \$42 per incidence, based on the mean o average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma attack is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study.
Acute Bronchitis	\$360	\$370	\$380	Assumes a 6-day episode, with daily value equal to the average of low and high values for related respiratory symptoms recommended in Neumann et al. (1994).

Table 4-11. Unit Values Used for Economic Valuation of Health Endpoints (1999\$) (continued)

	Central Estimate of Value Per Statistical Incidence				
Health Endpoint	1990 Income Level	2010 Income Level	2015 Income Level	- Derivation of Estimates	
Restricted Activity and Work/Sci	hool Loss Days				
Work Loss Days (WLDs)	Variable (national median =)			County-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5—to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.	
School Absence Days	\$75	\$75	\$75	Based on expected lost wages from parent staying home with child. Estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000 (U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 621). This median wage is \$551. Dividing by 5 gives an estimated median daily wage of \$103.	
				The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85% of \$103, or \$75.	
Worker Productivity	\$0.95 per worker per 10% change in ozone per day	\$0.95 per worker per 10% change in ozone per day	\$0.95 per worker per 10% change in ozone per day	Based on \$68—median daily earnings of workers in farming, forestry and fishing—from Table 621, Statistical Abstract of the United States ("Full-Time Wage and Salary Workers—Number and Earnings: 1985 to 2000") (Source of data in table: U.S. Bureau of Labor Statistics, Bulletin 2307 and Employment and Earnings, monthly).	
Minor Restricted Activity Days (MRADs)	\$51	\$53	\$54	Median WTP estimate to avoid one MRAD from Tolley et al. (1986).	

Table 4-11. Unit Values Used for Economic Valuation of Health Endpoints (1999\$) (continued)

end of the interquartile range from the Mrozek and Taylor (2002) meta-analysis. The \$10 million upper confidence limit represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. Because the majority of the studies in these meta-analyses are based on datasets from the early 1990s or previous decades, we continue to assume that the VSL estimates provided by those meta-analyses are in 1990 income equivalents. Future research might provide income-adjusted VSL values for individual studies that can be incorporated into the meta-analyses. This would allow for a more reliable base-year estimate for use in adjusting VSL for aggregate changes in income over time.

As indicated in the previous section on quantification of premature mortality benefits, we assumed for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the 20 years following exposure. To take this into account in the valuation of reductions in premature mortality, we applied an annual 3 percent discount rate to the value of premature mortality occurring in future years.¹⁷

The economics literature concerning the appropriate method for valuing reductions in premature mortality risk is still developing. The adoption of a value for the projected reduction in the risk of premature mortality is the subject of continuing discussion within the economics and public policy analysis community. Regardless of the theoretical economic considerations, EPA prefers not to draw distinctions in the monetary value assigned to the lives saved even if they differ in age, health status, socioeconomic status, gender, or other characteristic of the adult population.

Following the advice of the EEAC of the SAB, EPA currently uses the VSL approach in calculating the primary estimate of mortality benefits, because we believe this calculation provides the most reasonable single estimate of an individual's willingness to trade off money for reductions in mortality risk (EPA-SAB-EEAC-00-013, 2000). Although there are several differences between the labor market studies EPA uses to derive a VSL estimate and the PM air pollution context addressed here, those differences in the affected populations and the nature of the risks imply both upward and downward adjustments. Table 4-12 lists some of these differences and the expected effect on the VSL estimate for air pollution-related

¹⁷The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the federal government. EPA adopted a 3 percent discount rate for its base estimate in this case to reflect reliance on a "social rate of time preference" discounting concept. We have also calculated benefits and costs using a 7 percent rate consistent with an "opportunity cost of capital" concept to reflect the time value of resources directed to meet regulatory requirements. In this case, the benefit and cost estimates were not significantly affected by the choice of discount rate. Further discussion of this topic appears in EPA's *Guidelines for Preparing Economic Analyses* (EPA, 2000b).

Attribute	Expected Direction of Bias		
Age	Uncertain, perhaps overestimate		
Life Expectancy/Health Status	Uncertain, perhaps overestimate		
Attitudes Toward Risk	Underestimate		
Income	Uncertain		
Voluntary vs. Involuntary	Uncertain, perhaps underestimate		
Catastrophic vs. Protracted Death	Uncertain, perhaps underestimate		

Table 4-12. Expected Impact on Estimated Benefits of Premature Mortality Reductionsof Differences Between Factors Used in Developing Applied VSL and TheoreticallyAppropriate VSL

mortality. In the absence of a comprehensive and balanced set of adjustment factors, EPA believes it is reasonable to continue to use the \$5.5 million value while acknowledging the significant limitations and uncertainties in the available literature.

The SAB-EEAC has reviewed many potential VSL adjustments and the state of the economics literature. The SAB-EEAC advised EPA to "continue to use a wage-risk-based VSL as its primary estimate, including appropriate sensitivity analyses to reflect the uncertainty of these estimates," and that "the only risk characteristic for which adjustments to the VSL can be made is the timing of the risk" (EPA-SAB-EEAC-00-013, EPA, 2000). In developing our primary estimate of the benefits of premature mortality reductions, we have followed this advice and discounted over the lag period between exposure and premature mortality.

Uncertainties Specific to Premature Mortality Valuation. The economic benefits associated with premature mortality are the largest category of monetized benefits of the final CAIR. In addition, in prior analyses, EPA has identified valuation of mortality benefits as the largest contributor to the range of uncertainty in monetized benefits (see EPA [1999a]).¹⁸ Because of the uncertainty in estimates of the value of premature mortality avoidance, it is important to adequately characterize and understand the various types of

¹⁸This conclusion was based on a assessment of uncertainty based on statistical error in epidemiological effect estimates and economic valuation estimates. Additional sources of model error such as those examined in the pilot PM mortality expert elicitation may result in different conclusions about the relative contribution of sources of uncertainty.

economic approaches available for mortality valuation. Such an assessment also requires an understanding of how alternative valuation approaches reflect that some individuals may be more susceptible to air pollution-induced mortality or reflect differences in the nature of the risk presented by air pollution relative to the risks studied in the relevant economics literature.

The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. An ideal benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual's WTP to improve one's own chances of survival plus WTP to improve other individuals' survival rates. The ideal measure would also take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward and how individuals value these changes. Each individual's survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. For example, changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This probability shift will differ across individuals because survival curves depend on such characteristics as age, health state, and the current age to which the individual is likely to survive.

Although a survival curve approach provides a theoretically preferred method for valuing the benefits of reduced risk of premature mortality associated with reducing air pollution, the approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we value avoided premature mortality risk using the VSL approach.

Other uncertainties specific to premature mortality valuation include the following:

• Across-study variation: There is considerable uncertainty as to whether the available literature on VSL provides adequate estimates of the VSL saved by air pollution reduction. Although there is considerable variation in the analytical designs and data used in the existing literature, the majority of the studies involve the value of risks to a middle-aged working population. Most of the studies examine differences in wages of risky occupations, using a wage-hedonic approach. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the average WTP to

reduce the risk. The appropriateness of a distribution of WTP based on the current VSL literature for valuing the mortality-related benefits of reductions in air pollution concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on the extent to which the risks being valued are similar and the extent to which the subjects in the studies are similar to the population affected by changes in pollution concentrations.

- Level of risk reduction: The transferability of estimates of the VSL from the wage-risk studies to the context of the CAIR analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is \$50, but that the actual mortality risk reduction resulting from a given pollutant reduction is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of \$50 for a reduction of 1/100,000 implies a WTP of \$500 for a risk reduction of 1/10,000 (which is 10 times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the VSL does not depend on the particular amount of risk reduction being valued. This assumption has been shown to be reasonable provided the change in the risk being valued is within the range of risks evaluated in the underlying studies (Rowlatt et al., 1998).
- Voluntariness of risks evaluated: Although job-related mortality risks may differ in several ways from air pollution-related mortality risks, the most important difference may be that job-related risks are incurred voluntarily, or generally assumed to be, whereas air pollution-related risks are incurred involuntarily. Some evidence suggests that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily. If this is the case, WTP estimates based on wage-risk studies may understate WTP to reduce involuntarily incurred air pollution-related mortality risks.
- Sudden versus protracted death: A final important difference related to the nature of the risk may be that some workplace mortality risks tend to involve sudden, catastrophic events, whereas air pollution-related risks tend to involve longer periods of disease and suffering prior to death. Some evidence suggests that WTP to avoid a risk of a protracted death involving prolonged suffering and loss of dignity and personal control is greater than the WTP to avoid a risk (of identical magnitude) of sudden death. To the extent that the mortality risks addressed in this assessment are associated with longer periods of illness or greater pain and suffering than are the risks addressed in the valuation literature, the WTP measurements employed in the present analysis would reflect a downward bias.
- Self-selection and skill in avoiding risk: Recent research (Shogren and Stamland, 2002) suggests that VSL estimates based on hedonic wage studies may overstate

the average value of a risk reduction. This is based on the fact that the risk-wage trade-off revealed in hedonic studies reflects the preferences of the marginal worker (i.e., that worker who demands the highest compensation for his risk reduction). This worker must have either higher risk, lower risk tolerance, or both. However, the risk estimate used in hedonic studies is generally based on average risk, so the VSL may be upwardly biased because the wage differential and risk measures do not match.

4.1.5.4.2 Valuing Reductions in the Risk of Chronic Bronchitis. The best available estimate of WTP to avoid a case of CB comes from Viscusi et al. (1991). The Viscusi et al. study, however, describes a severe case of CB to the survey respondents. We therefore employ an estimate of WTP to avoid a pollution-related case of CB, based on adjusting the Viscusi et al. (1991) estimate of the WTP to avoid a severe case. This is done to account for the likelihood that an average case of pollution-related CB is not as severe. The adjustment is made by applying the elasticity of WTP with respect to severity reported in the Krupnick and Cropper (1992) study. Details of this adjustment procedure are provided in the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of CB in this analysis. The distribution incorporates uncertainty from three sources: the WTP to avoid a case of severe CB, as described by Viscusi et al.; the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi et al.); and the elasticity of WTP with respect to severity of the illness. Based on assumptions about the distributions of each of these three uncertain components, we derive a distribution of WTP to avoid a pollutionrelated case of CB by statistical uncertainty analysis techniques. The expected value (i.e., mean) of this distribution, which is about \$331,000 (2000\$), is taken as the central tendency estimate of WTP to avoid a PM-related case of CB.

4.1.5.4.3 Valuing Reductions in Nonfatal Myocardial Infarctions (Heart Attacks). The Agency has recently incorporated into its analyses the impact of air pollution on the expected number of nonfatal heart attacks, although it has examined the impact of reductions in other related cardiovascular endpoints. We were not able to identify a suitable WTP value for reductions in the risk of nonfatal heart attacks. Instead, we use a COI unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because the costs associated with a myocardial infarction extend beyond the initial event itself, we consider costs incurred over several years. Using age-specific annual lost earnings estimated by Cropper and Krupnick (1990) and a 3 percent discount

rate, we estimated a present discounted value in lost earnings (in 2000\$) over 5 years due to a myocardial infarction of \$8,774 for someone between the ages of 25 and 44, \$12,932 for someone between the ages of 45 and 54, and \$74,746 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2000\$) using a 7 percent discount rate are \$7,855, \$11,578, and \$66,920, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. As such, we do not include lost earnings in the cost estimates for these age groups.

We found three possible sources in the literature of estimates of the direct medical costs of myocardial infarction:

- Wittels et al. (1990) estimated expected total medical costs of myocardial • infarction over 5 years to be \$51,211 (in 1986\$) for people who were admitted to the hospital and survived hospitalization. (There does not appear to be any discounting used.) Wittels et al. was used to value coronary heart disease in the 812 Retrospective Analysis of the Clean Air Act. Using the CPI-U for medical care, the Wittels estimate is \$109,474 in year 2000\$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes, and prices (using "knowledgeable cardiologists" as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The authors note that the average length of hospitalization for acute myocardial infarction has decreased over time (from an average of 12.9 days in 1980 to an average of 11 days in 1983). Wittels et al. used 10 days as the average in their study. It is unclear how much further the length of stay for myocardial infarction may have decreased from 1983 to the present. The average length of stay for ICD code 410 (myocardial infarction) in the year-2000 Agency for Healthcare Research and Quality (AHRQ) HCUP database is 5.5 days. However, this may include patients who died in the hospital (not included among our nonfatal myocardial infarction cases), whose length of stay was therefore substantially shorter than it would be if they had not died.
- Eisenstein et al. (2001) estimated 10-year costs of \$44,663 in 1997\$, or \$49,651 in 2000\$ for myocardial infarction patients, using statistical prediction (regression) models to estimate inpatient costs. Only inpatient costs (physician fees and hospital costs) were included.
- Russell et al. (1998) estimated first-year direct medical costs of treating nonfatal myocardial infarction of \$15,540 (in 1995\$) and \$1,051 annually thereafter. Converting to year 2000\$, that would be \$23,353 for a 5-year period (without discounting) or \$29,568 for a 10-year period.

In summary, the three different studies provided significantly different values (see Table 4-13).

Study	Direct Medical Costs (2000\$)	Over an x-Year Period, for x =
Wittels et al. (1990)	\$109,474 ^a	5
Russell et al. (1998)	\$22,331 ^b	5
Eisenstein et al. (2001)	\$49,651 ^b	10
Russell et al. (1998)	\$27,242 ^b	10

 Table 4-13. Alternative Direct Medical Cost of Illness Estimates for Nonfatal Heart

 Attacks

^a Wittels et al. did not appear to discount costs incurred in future years.

^b Using a 3 percent discount rate.

As noted above, the estimates from these three studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used estimates for medical costs that similarly cover a 5-year period (i.e., estimates from Wittels et al. (1990) and Russell et al. (1998). We used a simple average of the two 5-year estimates, or \$65,902, and added it to the 5-year opportunity cost estimate. The resulting estimates are given in Table 4-14.

Table 4-14. Estimated Costs Over a 5-Year Period (in 2000\$) of a Nonfatal Myocardial Infarction

Age Group	Opportunity Cost	Medical Cost ^a	Total Cost
0–24	\$0	\$65,902	\$65,902
25–44	\$8,774 ^b	\$65,902	\$74,676
45–54	\$12,253 ^b	\$65,902	\$78,834
55–65	\$70,619 ^b	\$65,902	\$140,649
> 65	\$0	\$65,902	\$65,902

^a An average of the 5-year costs estimated by Wittels et al. (1990) and Russell et al. (1998).

^b From Cropper and Krupnick (1990), using a 3 percent discount rate.

4.1.5.4.4 Valuing Reductions in School Absence Days. School absences associated with exposure to ozone are likely to be due to respiratory-related symptoms and illnesses. Because the respiratory symptom and illness endpoints we are including are all PM-related rather than ozone-related, we do not have to be concerned about double-counting of benefits if we aggregate the benefits of avoiding ozone-related school absences with the benefits of avoiding PM-related respiratory symptoms and illnesses.

One possible approach to valuing a school absence is using a parental opportunity cost approach. This method requires two steps: estimate the probability that, if a school-aged child stays home from school, a parent will have to stay home from work to care for the child, and value the lost productivity at the person's wage. Using this method, we would estimate the proportion of families with school-age children in which both parents work and value a school loss day as the probability of a work loss day resulting from a school loss day (i.e., the proportion of households with school-age children in which both parents work) times some measure of lost wages (whatever measure we use to value work loss days). There are two significant problems with this method, however. First, it omits WTP to avoid the symptoms/illness that resulted in the school absence. Second, it effectively gives zero value to school absences that do not result in a work loss day (unless we derive an alternative estimate of the value of the parent's time for those cases in which the parent is not in the labor force). We are investigating approaches using WTP for avoid the symptoms/illnesses causing the absence. In the interim, we use the parental opportunity cost approach.

For the opportunity-cost approach, we make an explicit, conservative assumption that in married households with two working parents, the female parent will stay home with a sick child. From the U.S. Census Bureau, Statistical Abstract of the United States: 2001, we obtained 1) the numbers of single, married, and "other" (i.e., widowed, divorced, or separated) women with children in the workforce and 2) the rates of participation in the workforce of single, married, and "other" women with children. From these two sets of statistics, we inferred the numbers of single, married, and "other" women with children and the corresponding percentages. These percentages were used to calculate a weighted-average participation rate, as shown in Table 4-15.

Our estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000 (U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 621). This median wage is \$551. Dividing by 5 gives an estimated median daily wage of \$103.

	Number (in millions) in Labor Force (1)	Participation Rate (2)	Implied Total Number in Population (in millions) (3) = (1)/(2)	Implied Percent in Population (4)	Weighted Average Participation Rate [=sum (2)*(4) over rows]
Single	3.1	73.9%	4.19	11.84%	
Married	18.2	70.6%	25.78	72.79%	
Other ^b	4.5	82.7%	5.44	15.36%	
Total:			35.42		
					72.85%

 Table 4-15. Women with Children: Number and Percentage in the Labor Force, 2000, and Weighted Average Participation Rate^a

^a Data in columns (1) and (2) are from U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 577.

^b Widowed, divorced, or separated.

The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85 percent of \$103, or \$75.¹⁹ Of course, non-working parent time also has value. Determining that value is not straightforward. In a world with perfect labor markets, economic theory suggests that a non-working parent's time is worth at least (and generally exactly) that of a working parent. Otherwise, they would choose to work. Imperfect labor markets could imply a lower value of time, but still one above zero. Compounding the uncertainty about the value of time is uncertainty about the amount of time sacrificed by a non-working parent to care for a sick child. Since the value of a non-working parent's time is a function of the usual activities they perform, and caring for a child is presumably one of those activities, the crucial question is how much additional time is spent caring for a sick child during a school absence and what other activities are foregone as a result. Assuming a negligible reallocation of these activities

¹⁹In a recent article, Hall et al. (2003) use a similar methodology to derive a mid-estimate value per school absence day for California of between \$70 and \$81, depending on differences in incomes between three counties in California. Our national average estimate of \$75 per absence is consistent with these published values.

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and thus zero costs of a school absence to a household with a non-working parent provides a lower-bound on the benefits estimate for averting these impacts.

4.1.6 Human Welfare Impact Assessment

PM and ozone have numerous documented effects on environmental quality that affect human welfare. These welfare effects include direct damages to property, either through impacts on material structures or by soiling of surfaces, direct economic damages in the form of lost productivity of crops and trees, indirect damages through alteration of ecosystem functions, and indirect economic damages through the loss in value of recreational experiences or the existence value of important resources. EPA's Criteria Documents for PM and ozone list numerous physical and ecological effects known to be linked to ambient concentrations of these pollutants (EPA, 1996a; 1996b). This section describes individual effects and how we quantify and monetize them. These effects include changes in commercial crop and forest yields, visibility, and nitrogen deposition to estuaries.

4.1.6.1 Visibility Benefits

Changes in the level of ambient PM caused by the reduction in emissions from CAIR will change the level of visibility in much of the Eastern United States. Visibility directly affects people's enjoyment of a variety of daily activities. Individuals value visibility both in the places they live and work, in the places they travel to for recreational purposes, and at sites of unique public value, such as the Great Smokey Mountains National Park. This section discusses the measurement of the economic benefits of improved visibility.

It is difficult to quantitatively define a visibility endpoint that can be used for valuation. Increases in PM concentrations cause increases in light extinction, a measure of how much the components of the atmosphere absorb light. More light absorption means that the clarity of visual images and visual range is reduced, *ceteris paribus*. Light absorption is a variable that can be accurately measured. Sisler (1996) created a unitless measure of visibility, the *deciview*, based directly on the degree of measured light absorption. Deciviews are standardized for a reference distance in such a way that one deciview corresponds to a change of about 10 percent in available light. Sisler characterized a change in light extinction of one deciview as "a small but perceptible scenic change under many

circumstances." Air quality models were used to predict the change in visibility, measured in deciviews, of the areas affected by the control options.²⁰

EPA considers benefits from two categories of visibility changes: residential visibility and recreational visibility. In both cases economic benefits are believed to consist of use values and nonuse values. Use values include the aesthetic benefits of better visibility, improved road and air safety, and enhanced recreation in activities like hunting and birdwatching. Nonuse values are based on people's beliefs that the environment ought to exist free of human-induced haze. Nonuse values may be more important for recreational areas, particularly national parks and monuments.

Residential visibility benefits are those that occur from visibility changes in urban, suburban, and rural areas and also in recreational areas not listed as federal Class I areas.²¹ For the purposes of this analysis, recreational visibility improvements are defined as those that occur specifically in federal Class I areas. A key distinction between recreational and residential benefits is that only those people living in residential areas are assumed to receive benefits from residential visibility, while all households in the United States are assumed to derive some benefit from improvements in Class I areas. Values are assumed to be higher if the Class I area is located close to their home.²²

Only two existing studies provide defensible monetary estimates of the value of visibility changes. One is a study on residential visibility conducted in 1990 (McClelland et al., 1993) and the other is a 1988 survey on recreational visibility value (Chestnut and Rowe, 1990a; 1990b). Although there are a number of other studies in the literature, they were conducted in the early 1980s and did not use methods that are considered defensible by current standards. Both the Chestnut and Rowe and McClelland et al. studies use the CV method. There has been a great deal of controversy and significant development of both

²⁰A change of less than 10 percent in the light extinction budget represents a measurable improvement in visibility but may not be perceptible to the eye in many cases. Some of the average regional changes in visibility are less than one deciview (i.e., less than 10 percent of the light extinction budget) and thus less than perceptible. However, this does not mean that these changes are not real or significant. Our assumption is then that individuals can place values on changes in visibility that may not be perceptible. This is quite plausible if individuals are aware that many regulations lead to small improvements in visibility that, when considered together, amount to perceptible changes in visibility.

²¹The Clean Air Act designates 156 national parks and wilderness areas as Class I areas for visibility protection.

²²For details of the visibility estimates discussed in this chapter, please refer to the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

theoretical and empirical knowledge about how to conduct CV surveys in the past decade. In EPA's judgment, the Chestnut and Rowe study contains many of the elements of a valid CV study and is sufficiently reliable to serve as the basis for monetary estimates of the benefits of visibility changes in recreational areas.²³ This study serves as an essential input to our estimates of the benefits of recreational visibility improvements in the primary benefits estimates. Consistent with SAB advice, EPA has designated the McClelland et al. study as significantly less reliable for regulatory benefit-cost analysis, although it does provide useful estimates on the order of magnitude of residential visibility benefits (EPA-SAB-COUNCIL-ADV-00-002, 1999). Residential visibility benefits are not calculated for this analysis.

The Chestnut and Rowe study measured the demand for visibility in Class I areas managed by the National Park Service (NPS) in three broad regions of the country: California, the Southwest, and the Southeast. Respondents in five states were asked about their WTP to protect national parks or NPS-managed wilderness areas within a particular region. The survey used photographs reflecting different visibility levels in the specified recreational areas. The visibility levels in these photographs were later converted to deciviews for the current analysis. The survey data collected were used to estimate a WTP equation for improved visibility. In addition to the visibility change variable, the estimating equation also included household income as an explanatory variable.

The Chestnut and Rowe study did not measure values for visibility improvement in Class I areas outside the three regions. Their study covered 86 of the 156 Class I areas in the United States. We can infer the value of visibility changes in the other Class I areas by transferring values of visibility changes at Class I areas in the study regions. A complete description of the benefits transfer method used to infer values for visibility changes in Class I areas outside the study regions is provided in the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

The Chestnut and Rowe study (Chestnut and Rowe, 1990a; 1990b), although representing the best available estimates, has a number of limitations. These include the following:

²³An SAB advisory letter indicates that "many members of the Council believe that the Chestnut and Rowe study is the best available" (EPA-SAB-COUNCIL-ADV-00-002, 1999, p. 13). However, the committee did not formally approve use of these estimates because of concerns about the peer-reviewed status of the study. EPA believes the study has received adequate review and has been cited in numerous peer-reviewed publications (Chestnut and Dennis, 1997).

- The age of the study (late 1980s) will increase the uncertainty about the correspondence of the estimated values to those that might be provided by current or future populations.
- The survey focused only on populations in five states, so the application of the estimated values to populations outside those states requires that preferences of populations in the five surveyed states be similar to those of nonsurveyed states.
- There is an inherent difficulty in separating values expressed for visibility improvements from an overall value for improved air quality. The Chestnut and Rowe study attempted to control for this by informing respondents that "other households are being asked about visibility, human health, and vegetation protections in urban areas and at national parks in other regions." However, most of the respondents did not feel that they were able to segregate visibility at national parks entirely from residential visibility and health effects.
- It is not clear exactly what visibility improvements the respondents to the Chestnut and Rowe survey were valuing. For the purpose of the benefits analysis for this rule, EPA assumed that respondents provided values for changes in annual average visibility. Because most policies will result in a shift in the distribution of visibility (usually affecting the worst days more than the best days), the annual average may not be the most relevant metric for policy analysis.
- The WTP question asked about changes in average visibility. However, the survey respondents were shown photographs of only summertime conditions, when visibility is generally at its worst. It is possible that the respondents believed those visibility conditions held year-round, in which case they would have been valuing much larger overall improvements in visibility than what otherwise would be the case.
- The survey did not include reminders of possible substitutes (e.g., visibility at other parks) or budget constraints. These reminders are considered to be best practice for stated preference surveys.
- The Chestnut and Rowe survey focused on visibility improvements in and around national parks and wilderness areas. The survey also focused on visibility improvements of national parks in the southwest United States. Given that national parks and wilderness areas exhibit unique characteristics, it is not clear whether the WTP estimate obtained from Chestnut and Rowe can be transferred to other national parks and wilderness areas, without introducing additional uncertainty.

In general, the survey design and implementation reflect the period in which the survey was conducted. Since that time, many improvements to the stated preference methodology have

been developed. As future survey efforts are completed, EPA will incorporate values for visibility improvements reflecting the improved survey designs.

The estimated relationship from the Chestnut and Rowe study is only directly applicable to the populations represented by survey respondents. EPA used benefits transfer methodology to extrapolate these results to the population affected by the final CAIR. A general WTP equation for improved visibility (measured in deciviews) was developed as a function of the baseline level of visibility, the magnitude of the visibility improvement, and household income. The behavioral parameters of this equation were taken from analysis of the Chestnut and Rowe data. These parameters were used to calibrate WTP for the visibility changes resulting from CAIR. The method for developing calibrated WTP functions is based on the approach developed by Smith et al. (2002). Available evidence indicates that households are willing to pay more for a given visibility improvement as their income increases (Chestnut, 1997). The benefits estimates here incorporate Chestnut's estimate that a 1 percent increase in income is associated with a 0.9 percent increase in WTP for a given change in visibility.

Using the methodology outlined above, EPA estimates that the total WTP for the visibility improvements in Southeastern Class I areas brought about by CAIR is \$1.14 billion in 2010 and \$1.78 billion in 2015. This value includes the value to households living in the same state as the Class I area as well as values for all households in the United States living outside the state containing the Class I area, and the value accounts for growth in real income.

The benefits resulting from visibility improvements in Southeastern Class I areas under the final CAIR are presented in Figure 4-2. This figure presents these benefits both in terms of the total benefits modeled for each of the Class I areas (i.e., the "Park Benefits" map) and the benefits realized by the populations in each of the 48 contiguous states (i.e., the "State Benefits" map). The latter results reflect the WTP of state residents for visibility improvements occurring in Class I areas in the Southeastern United States.

One major source of uncertainty for the visibility benefits estimate is the benefits transfer process used. Judgments used to choose the functional form and key parameters of the estimating equation for WTP for the affected population could have significant effects on the size of the estimates. Assumptions about how individuals respond to changes in visibility that are either very small or outside the range covered in the Chestnut and Rowe study could also affect the results.

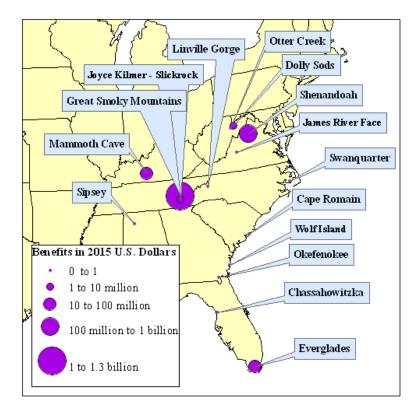


Figure 4-2. CAIR Final Rule Visibility Improvements in Class I Areas in the Southeast

4.1.6.2 Agricultural, Forestry, and Other Vegetation-Related Benefits

The Ozone Criteria Document notes that ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant (EPA, 1996a, page 5-11). Changes in ground-level ozone resulting from the control options are expected to affect crop and forest yields throughout the affected area.

Well-developed techniques exist to provide monetary estimates of these benefits to agricultural producers and to consumers. These techniques use models of planting decisions, yield response functions, and the supply of and demand for agricultural products. The resulting welfare measures are based on predicted changes in market prices and production costs. Models also exist to measure benefits to silvicultural producers and consumers. However, these models have not been adapted for use in analyzing ozone-related forest impacts. Because of resource limitations, we are unable to provide agricultural or benefits estimates for the final CAIR rule.

4.1.6.2.1 Agricultural Benefits. Laboratory and field experiments have shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g.,

lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN), examined 15 species and numerous cultivars. The NCLAN results show that several economically important crop species are sensitive to ozone levels typical of those found in the United States (EPA, 1996a). In addition, economic studies have shown a relationship between observed ozone levels and crop yields (Garcia et al., 1986).

4.1.6.2.2 Forestry Benefits. Ozone also has been shown conclusively to cause discernible injury to forest trees (EPA, 1996a; Fox and Mickler, 1996). In our previous analysis of the HD Engine/Diesel Fuel rule, we were able to quantify the effects of changes in ozone concentrations on tree growth for a limited set of species. Because of resource limitations, we were not able to quantify such impacts for this analysis.

4.1.6.2.3 Other Vegetation Effects. An additional welfare benefit expected to accrue as a result of reductions in ambient ozone concentrations in the United States is the economic value the public receives from reduced aesthetic injury to forests. There is sufficient scientific information available to reliably establish that ambient ozone levels cause visible injury to foliage and impair the growth of some sensitive plant species (EPA, 1996a). However, present analytic tools and resources preclude EPA from quantifying the benefits of improved forest aesthetics.

Urban ornamentals (floriculture and nursery crops) represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to affect large economic sectors. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative economic benefits analysis has been conducted. The farm production value of ornamental crops was estimated at over \$14 billion in 2003 (USDA, 2004). This is therefore a potentially important welfare effects category. However, information and valuation methods are not available to allow for plausible estimates of the percentage of these expenditures that may be related to impacts associated with ozone exposure.

The EGU program, by reducing NO_x emissions, will also reduce nitrogen deposition on agricultural land and forests. There is some evidence that nitrogen deposition may have positive effects on agricultural output through passive fertilization. Holding all other factors constant, farmers' use of purchased fertilizers or manure may increase as deposited nitrogen is reduced. Estimates of the potential value of this possible increase in the use of purchased fertilizers are not available, but it is likely that the overall value is very small relative to other health and welfare effects. The share of nitrogen requirements provided by this deposition is small, and the marginal cost of providing this nitrogen from alternative sources is quite low. In some areas, agricultural lands suffer from nitrogen oversaturation due to an abundance of on-farm nitrogen production, primarily from animal manure. In these areas, reductions in atmospheric deposition of nitrogen represent additional agricultural benefits.

Information on the effects of changes in passive nitrogen deposition on forests and other terrestrial ecosystems is very limited. The multiplicity of factors affecting forests, including other potential stressors such as ozone, and limiting factors such as moisture and other nutrients, confound assessments of marginal changes in any one stressor or nutrient in forest ecosystems. However, reductions in the deposition of nitrogen could have negative effects on forest and vegetation growth in ecosystems where nitrogen is a limiting factor (EPA, 1993).

On the other hand, there is evidence that forest ecosystems in some areas of the United States are nitrogen saturated (EPA, 1993). Once saturation is reached, adverse effects of additional nitrogen begin to occur such as soil acidification, which can lead to leaching of nutrients needed for plant growth and mobilization of harmful elements such as aluminum. Increased soil acidification is also linked to higher amounts of acidic runoff to streams and lakes and leaching of harmful elements into aquatic ecosystems.

4.1.6.3 Benefits from Reductions in Materials Damage

The control options that we modeled are expected to produce economic benefits in the form of reduced materials damage. There are two important categories of these benefits. Household soiling refers to the accumulation of dirt, dust, and ash on exposed surfaces. Criteria pollutants also have corrosive effects on commercial/industrial buildings and structures of cultural and historical significance. The effects on historic buildings and outdoor works of art are of particular concern because of the uniqueness and irreplaceability of many of these objects.

Previous EPA benefits analyses have been able to provide quantitative estimates of household soiling damage. Consistent with SAB advice, we determined that the existing data (based on consumer expenditures from the early 1970s) are too out of date to provide a reliable estimate of current household soiling damages (EPA-SAB-COUNCIL-ADV-98-003, 1998).

EPA is unable to estimate any benefits to commercial and industrial entities from reduced materials damage. Nor is EPA able to estimate the benefits of reductions in PM-

related damage to historic buildings and outdoor works of art. Existing studies of damage to this latter category in Sweden (Grosclaude and Soguel, 1994) indicate that these benefits could be an order of magnitude larger than household soiling benefits.

4.1.6.4 Benefits from Reduced Ecosystem Damage

The effects of air pollution on the health and stability of ecosystems are potentially very important but are at present poorly understood and difficult to measure. The reductions in NO_x caused by the final rule could produce significant benefits. Excess nutrient loads, especially of nitrogen, cause a variety of adverse consequences to the health of estuarine and coastal waters. These effects include toxic and/or noxious algal blooms such as brown and red tides, low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters, the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats, and fundamental shifts in phytoplankton community structure (Bricker et al., 1999).

Direct functions relating changes in nitrogen loadings to changes in estuarine benefits are not available. The preferred WTP-based measure of benefits depends on the availability of these functions and on estimates of the value of environmental responses. Because neither appropriate functions nor sufficient information to estimate the marginal value of changes in water quality exist at present, calculation of a WTP measure is not possible.

If better models of ecological effects can be defined, EPA believes that progress can be made in estimating WTP measures for ecosystem functions. These estimates would be superior to avoided cost estimates in placing economic values on the welfare changes associated with air pollution damage to ecosystem health. For example, if nitrogen or sulfate loadings can be linked to measurable and definable changes in fish populations or definable indexes of biodiversity, then CV studies can be designed to elicit individuals' WTP for changes in these effects. This is an important area for further research and analysis and will require close collaboration among air quality modelers, natural scientists, and economists.

4.2 Benefits Analysis—Results

Applying the impact and valuation functions described previously in this chapter to the estimated changes in ozone and PM yields estimates of the changes in physical damages (e.g., premature mortalities, cases, admissions, change in light extinction) and the associated monetary values for those changes. Estimates of physical health impacts are presented in Table 4-16. Monetized values for both health and welfare endpoints are presented in Table 4-17, along with total aggregate monetized benefits. All of the monetary benefits are in constant-year 1999 dollars.

	2010	2015
Health Effect	Incidence	e Reduction
PM-Related Endpoints		
Premature Mortality ^{b,c}		
Adult, age 30 and over	13,000	17,000
Infant, age <1 year	29	36
Chronic bronchitis (adult, age 26 and over)	6,900	8,700
Nonfatal myocardial infarction (adults, age 18 and older)	17,000	22,000
Hospital admissions—respiratory (all ages) ^d	4,300	5,500
Hospital admissions—cardiovascular (adults, age >18) ^e	3,800	5,000
Emergency room visits for asthma (age 18 years and younger)	10,000	13,000
Acute bronchitis (children, age 8-12)	16,000	19,000
Lower respiratory symptoms (children, age 7-14)	190,000	230,000
Upper respiratory symptoms (asthmatic children, age 9–18)	150,000	180,000
Asthma exacerbation (asthmatic children, age 6-18)	240,000	290,000
Work loss days (adults, age 18–65)	1,400,000	1,700,000
Minor restricted-activity days (adults, age 18-65)	8,100,000	9,900,000
Ozone-Related Endpoints		
Hospital admissions—respiratory causes (adult, 65 and older) ^f	610	1,700
Hospital admissions-respiratory causes (children, under 2)	380	1,100
Emergency room visit for asthma (all ages)	100	280
Minor restricted-activity days (adults, age 18-65)	280,000	690,000
School absence days	180,000	510,000

 Table 4-16. Clean Air Interstate Rule: Estimated Reduction in Incidence of Adverse Health Effects^a

^a Incidences are rounded to two significant digits. These estimates represent benefits for CAIR Nationwide for the final CAIR program inclusive of the proposal to include SO_2 and annual NO_x controls for New Jersey and Delaware. Note these estimates may be slightly overstated due to the inclusion of SO_2 and annual NO_x controls for Arkansas.

^b Premature mortality benefits associated with ozone are not addressed in the primary analysis.

^c PM premature mortality impacts for adults are based on application of the effect estimate derived from the Pope et al (2002) cohort study. Infant premature mortality based upon studies by Woodruff, et al 1997.

^d Respiratory hospital admissions for PM include admissions for COPD, pneumonia, and asthma.

^e Cardiovascular hospital admissions for PM include total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

^f Respiratory hospital admissions for ozone include admissions for total respiratory and subcategories for COPD and pneumonia.

 Table 4-17. Estimated Monetary Value in Reductions in Incidence of Health and

 Welfare Effects (in millions of 1999\$)^{a,b}

		2010	2015
Health Effect	Pollutant	Estimated Value of Reductions	
Premature mortality ^{c,d}			
Adult, age 30 and over			
3% discount rate	PM _{2.5}	\$67,300	\$92,800
7% discount rate		\$56,600	\$78,100
Infant, < 1 year		\$168	\$222
Chronic bronchitis (adults, 26 and over)	PM _{2.5}	\$2,520	\$3,340
Non-fatal acute myocardial infarctions			
3% discount rate	PM _{2.5}	\$1,420	\$1,850
7% discount rate		\$1,370	\$1,790
Hospital admissions for respiratory causes	PM _{2.5} , O ₃	\$45.2	\$78.9
Hospital admissions for cardiovascular causes	PM _{2.5}	\$80.7	\$105
Emergency room visits for asthma	PM _{2.5} ,O ₃	\$2.84	\$3.56
Acute bronchitis (children, age 8-12)	PM _{2.5}	\$5.63	\$7.06
Lower respiratory symptoms (children, 7–14)	PM _{2.5}	\$2.98	\$3.74
Upper respiratory symptoms (asthma, 9–11)	PM _{2.5}	\$3.80	\$4.77
Asthma exacerbations	PM _{2.5}	\$10.3	\$12.7
Work loss days	PM _{2.5} ,	\$180	\$219
Minor restricted-activity days (MRADs)	PM _{2.5} ,O ₃	\$422	\$543
School absence days	O_3	\$12.9	\$36.4
Worker productivity (outdoor workers, 18–65)	O_3	\$7.66	\$19.9
Recreational visibility, 81 Class I areas	PM _{2.5}	\$1,140	\$1,780
Monetized Total ^e			
Base Estimate:	PM _{2.5} ,O ₃		
3% discount rate		\$73,300 + B	\$101,000 + B
7% discount rate		\$62,600 + B	\$86,300 + B

^a Monetary benefits are rounded to three significant digits for ease of presentation and computation. Benefits in this table are nationwide (with the exception of visibility and ozone) and are associated with NO_x and SO₂ reductions at EGU sources. Ozone benefits relate to the eastern United States. Visibility benefits relate to Class I areas in the southeastern United States. These estimates relate to the final CAIR program inclusive of the proposal to include SO₂ and annual NO_x controls for New Jersey and Delaware. Note that these estimates may be slightly overstated due to the inclusion of SO₂ and annual NO_x controls for Arkansas in the modeling.

^b Monetary benefits adjusted to account for growth in real GDP per capita between 1990and the analysis year (2010 or 2015)

^c Valuation assumes discounting over the SAB recommended 20 year segmented lag structure described earlier. Results reflect the use of 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000b; OMB, 2003).

^d Adult premature mortality estimates based upon studies by Pope, et al 2002. Infant premature mortality based upon Woodruff et al 1997.

^e B represents the monetary value of health and welfare benefits and disbenefits not monetized. A detailed listing is provided in Table 4-2.

Not all known PM- and ozone-related health and welfare effects could be quantified or monetized. The monetized value of these unquantified effects is represented by adding an unknown "B" to the aggregate total. The estimate of total monetized health benefits is thus equal to the subset of monetized PM- and ozone-related health and welfare benefits plus B, the sum of the nonmonetized health and welfare benefits.

Total monetized benefits are dominated by benefits of mortality risk reductions. The primary analysis estimate projects that the final rule will result in 13,000 avoided premature deaths annually in 2010 and 17,000 avoided premature deaths annually in 2015. The increase in annual benefits from 2010 to 2015 reflects additional emission reductions from the standards, as well as increases in total population and the average age (and thus baseline mortality risk) of the population. Note that unaccounted for changes in baseline mortality rates over time may lead to reductions in the estimated number of avoided premature mortalities.

Our estimate of total monetized benefits in 2010 for the final rule is \$73.3 billion using a 3 percent discount rate and \$62.6 billion using a 7 percent discount rate. In 2015, the monetized benefits are estimated at \$101 billion using a 3 percent discount rate and \$86.3 billion using a 7 percent discount rate. Health benefits account for 98 percent of total benefits, in part because we are unable to quantify most of the nonhealth benefits. These unquantified benefits may be substantial and could exceed the costs of the rule, although the magnitude of these benefits is highly uncertain. The monetized benefit associated with reductions in the risk of premature mortality, which accounts for \$67.3 billion in 2010 and \$92.8 billion in 2015, is over 90 percent of total monetized health benefits. The next largest benefit is for reductions in chronic illness (CB and nonfatal heart attacks), although this value is more than an order of magnitude lower than for premature mortality. Hospital admissions for respiratory and cardiovascular causes, visibility, MRADs, work loss days, school absence days, and worker productivity account for the majority of the remaining benefits. The remaining categories each account for a small percentage of total benefit; however, they represent a large number of avoided incidences affecting many individuals. A comparison of the incidence table to the monetary benefits table reveals that there is not always a close correspondence between the number of incidences avoided for a given endpoint and the monetary value associated with that endpoint. For example, there are almost 100 times more work loss days than premature mortalities, yet work loss days account for only a very small fraction of total monetized benefits. This reflects the fact that many of the less severe health effects, while more common, are valued at a lower level than the more severe health effects. Also, some effects, such as hospital admissions, are valued using a

proxy measure of WTP. As such, the true value of these effects may be higher than that reported in Table 4-16.

Ozone benefits are in aggregate positive for the nation. However, because ozone increases occur during certain hours of the day in some urban areas, there is a dampening of overall ozone benefits in both 2010 and 2015, although the net incidence and benefits estimates for all health effects categories are net positive. Overall, ozone benefits are low relative to PM benefits for similar endpoint categories because of the increases in ozone concentrations during some hours of some days in certain urban areas.

4.2.1 Potential Benefits of the New Jersey and Delaware Proposal

The benefit estimate results presented reflect benefits for the final CAIR program inclusive of the New Jersey and Delaware proposal. Air quality modeling was not conducted for the New Jersey and Delaware proposal. For this reason, an analysis of the potential benefits for the New Jersey and Delaware proposal could not be completed with any degree of specificity. However based on the air quality modeling results for the CAIR, we make rough estimates of the benefits that might occur with this proposal. Including New Jersey and Delaware in the CAIR program would result in additional reductions of SO₂ and NO_x emissions. We estimate that approximately \$630 million of the total annual CAIR program benefits previously discussed could be attributable to annual SO₂ and NO_x controls for New Jersey and Delaware in 2010 and approximately \$1.1 billion could be attributable to annual SO₂ and NO_x controls for New Jersey and Delaware in 2015.

4.3 Probabilistic Analysis of Uncertainty in the Benefits Estimates

The recent NRC report on estimating public health benefits of air pollution regulations recommended that EPA begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabilistic, multiple-source uncertainty analyses (NRC, 2002). The probability distributions required for these analyses should be based on available data and expert judgment. The NRC also recommended that EPA use both internal and external experts as needed, in each case identifying those experts whose judgments are used and the rationales and empirical bases for their judgments.

As part of an overall program to improve the Agency's characterization of uncertainties in health benefits analyses, this section describes EPA's initial efforts to address uncertainties associated with the PM mortality C-R relationship and valuation. Similar to our approach in the Nonroad Diesel RIA, for the CAIR, we present two types of probabilistic approaches. The first approach generates a distribution of benefits based on the sampling error and variability in the underlying health and economic valuation studies used in the benefits modeling framework. The second approach uses the results from a pilot expert elicitation project designed to characterize key aspects of uncertainty in the ambient $PM_{2.5}$ /mortality relationship. Both approaches provide insights into the likelihood of different outcomes and about the state of knowledge regarding the benefits estimates. Both approaches have different strengths and weaknesses, that are summarized below.

We provide likelihood distributions both for the total dollar benefits estimate and for the incidence of premature mortality to show the uncertainty described by each expert's judgment relative to the range of uncertainty associated with the standard error in the Pope et al. (2002) study. The uncertainty about the total dollar benefit associated with any single endpoint combines the uncertainties from two sources—the C-R relationship and the valuation—and is estimated with a Monte Carlo method.²⁴ Our estimates of the likelihood distributions for total benefits should be viewed within the context of the wide range of sources of uncertainty that we have not incorporated, including uncertainty in emissions, air quality, and baseline health effect incidence rates.

In benefit analyses of air pollution regulations conducted to date, the estimated impact of reductions in premature mortality has accounted for 85 to 95 percent of total benefits. Therefore, in characterizing the uncertainty related to the estimates of total benefits it is particularly important to attempt to characterize the uncertainties associated with this endpoint. We conducted two different Monte Carlo analyses, one based on the distribution of reductions in premature mortality characterized by the mean effect estimate and standard error from the Pope et al. (2002) study (our primary estimate), and one based on the results from a pilot expert elicitation project (IEc, 2004). The Pope et al. study is described earlier in this chapter. A detailed discussion of the pilot expert elicitation project is provided in Appendix B. We summarize several key points about the project below.

As a first step in addressing the NRC recommendations regarding expert elicitation, EPA, in collaboration with OMB, conducted a pilot expert elicitation to characterize uncertainties in the relationship between ambient $PM_{2.5}$ and mortality. This pilot was designed to provide EPA with an opportunity to improve its understanding of the design and

²⁴In each iteration of the Monte Carlo procedure, a value is randomly drawn from the incidence distribution, and a value is randomly drawn from the unit dollar value distribution. The total dollar benefit for that iteration is the product of the two. If this is repeated for many (e.g., thousands of) iterations, the distribution of total dollar benefits associated with the endpoint is generated. For details on the specific Monte Carlo approach we used, see Appendix B.

application of expert elicitation methods to economic benefits analysis, to lay the groundwork for a more comprehensive elicitation, and to provide more information about the uncertainty in the $PM_{2.5}$ -mortality relationship in the context of the Nonroad Diesel RIA and similar analyses conducted in the near term.

The pilot project elicited the judgments of five experts in the PM health sciences, all members of at least one of two recent National Academy of Sciences scientific committees focused on particulate matter. The specific process used to select experts is summarized in Appendix B and detailed in the technical report describing the elicitation (IEc, 2004) along with additional information about the experts' affiliations and fields of expertise. The responses of each expert to questions enumerated in the elicitation protocol provide the inputs for developing distributions of mortality benefits.

The elicitation approach tested in the pilot was peer reviewed by four experts (Mansfield, 2004) and generally received favorable comments, including

- the pilot project followed "best practices" for expert elicitation and was well documented,
- the elicitation was well conducted and is an appropriate technique for characterizing uncertainty,
- experts chosen are well known and respected and represent a range of views, and
- the expert selection process/protocol was very good.

The peer-review report also provides recommendations on how to improve the process for a more comprehensive expert elicitation addressing the PM mortality C-R relationship. Specifically, the peer-review recommendations included

- holding a pre-elicitation workshop to ensure that all experts are properly motivated and conditioned (i.e., on the same footing) before the interviews and to allow for information sharing prior to the elicitation;
- allowing for some form of communication following the individual interviews to allow review of the experts' responses and allow them to adjust their estimates if necessary—one way to accomplish this is through a post-elicitation workshop; and
- changing the encoding process to ensure that extreme values (upper and lower ranges) are collected prior to judgments on central tendency. This sequencing would avoid anchoring or adjustment heuristics associated with biased estimates of uncertainty.

In addition, although not listed explicitly as a criticism, some of the peer reviewers noted the small number of experts participating in the pilot and suggested a larger number of experts should be used in EPA's next elicitation.

The peer reviewers also offered varying comments on the methods for combining the results of the pilot elicitation. Several of the reviewers preferred that the expert opinions not be combined or stated that they knew of no agreed-upon method for combining results from expert elicitations. They stated that presenting each expert's response independently allows for differences in the individual distributions to be recognized. Two of the reviewers indicated that they were reasonably comfortable with the method used in this study to combine the results, while the other two reviewers offered comments on the combined result of the elicitation. One reviewer stated that the combined distributions do not adequately capture the opinions of individual experts but rather average them out. He states that it is possible in such cases that the combined judgments may generate results that none of the experts would agree on. Another reviewer stated that expert elicitation studies typically do not combine judgments, but if one were to combine them, he recommended that the response of each be maintained independently from the other experts and run through the benefits model completely prior to combining the results.

For more details regarding the peer-review comments, see Appendix B. The full peerreview report is also available at www.epa.gov/ttn/ecas/benefits.html.

The distributions of all other, nonmortality health endpoints are characterized by the reported mean and standard deviations from the epidemiology literature. Details on the distributions used for individual health endpoints are provided in Appendix B. We are unable at this time to characterize the uncertainty in the estimate of benefits of improvements in visibility at Class I areas. As such, we treat the visibility benefits as fixed and add them to all percentiles of the health benefits distribution. Given this unequal treatment of endpoints, it is likely that these distributions do not capture the full range of benefits, and in fact are likely to understate the uncertainty, especially on the high end of the range due to omission of potentially significant benefit categories. We include them here primarily as an illustration of the impacts of using probabilistic (expert elicitation and statistical error-based) distributions for premature mortality associated with $PM_{2.5}$ compared with EPA's traditional approach.

Figure 4-3 presents box plots of the distributions of the reduction in $PM_{2.5}$ -related premature mortality based on the C-R distributions provided by each expert, as well as that for our primary estimate, based on the statistical error associated with Pope et al. (2002).

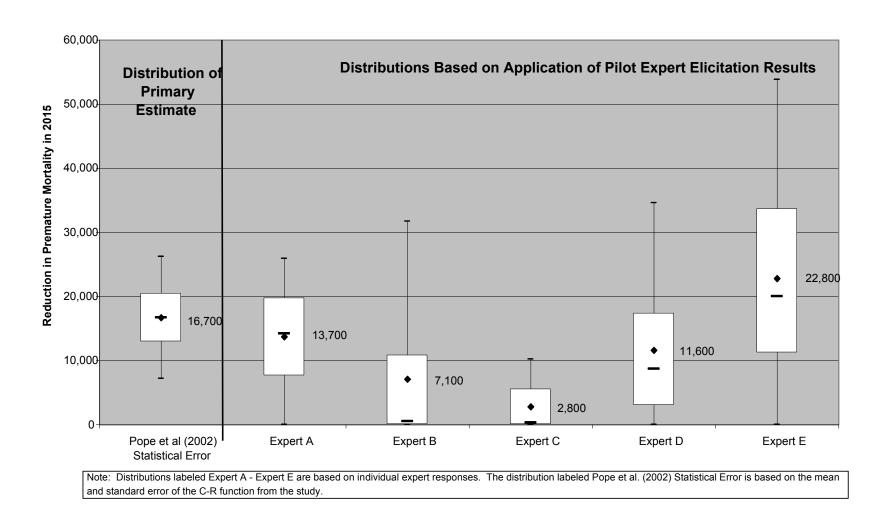


Figure 4-3. Results of Illustrative Application of Pilot Expert Elicitation: Annual Reductions in Premature Mortality in 2015 Associated with the Clean Air Interstate Rule

The distributions are depicted as box plots with the diamond symbol (\blacklozenge) showing the mean, the dash (-) showing the median (50th percentile), the box defining the interquartile range (bounded by the 25th and 75th percentiles), and the whiskers defining the 90 percent confidence interval (bounded by the 5th and 95th percentiles of the distribution). Our primary estimate based on the Pope et al. (2002) study shows that the average number of premature deaths avoided in 2015 is 16,700. This is higher than four of the experts and lower than one expert and falls within the uncertainty bounds of all but one expert. The figure shows that the average annual number of premature deaths avoided in 2015 ranges from approximately 2,800 (based on the judgments of Expert C) to 22,800 (based on the judgments of Expert E). The medians span zero to 20,000, with the zero value due to the high threshold associated with one of the expert's distributions. The statistical uncertainty bounds of all of the estimates, including the Pope et al.-based distribution, overlap. Although the uncertainty bounds for each expert include zero, and some distributions have significant percentiles at zero, all of the distributions have a positive mean estimate.

Figure 4-4 presents box plots of the distributions of monetized benefits of reductions in premature mortality associated with use of the Pope et al. (2002) and expert-based mortality incidence distributions. Our primary estimate based on the Pope et al. (2002) study shows that the mean annual benefit is roughly \$93 billion. Mean annual benefits for each expert elicited during the pilot expert elicitation range from approximately \$16 billion (based on judgments of Expert C) to \$130 billion (based on the judgments of Expert E). Impacts on the distribution of total benefits (including visibility and non-mortality health benefits) are discussed in Appendix B.

The uncertainty estimates based on statistical error have the strength of presenting a statistical measure of the uncertainty in the underlying studies serving as the basis for the estimates used in the analysis. However, this approach captures only a limited portion of the uncertainty about the parameters. The 5th and 95th percentile points of the distributions are based on statistical error and cross-study variability and provide some insight into how uncertain our estimate is with regard to those sources of uncertainty. However, it does not capture other sources of uncertainty regarding the model specification and other inputs to the model, including emissions, air quality, and aspects of the health science not captured in the studies, such as the likelihood that PM is causally related to premature mortality and other serious health effects.

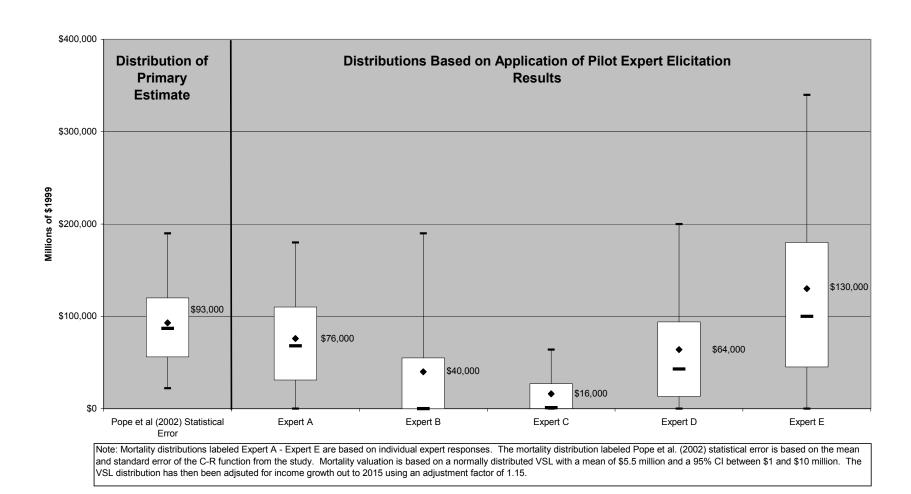


Figure 4-4. Results of Illustrative Application of Pilot Expert Elicitation: Dollar Value of Annual Reductions in Premature Mortality in 2015 Associated with the Clean Air Interstate Rule

4.4 Discussion

This analysis has estimated the health and welfare benefits of reductions in ambient concentrations of particulate matter and ozone resulting from reduced emissions of NO_x and SO_2 from affected EGUs. The result suggests there will be significant health and welfare benefits arising from regulating emissions from EGUs in the United States. Our estimate that 17,000 premature mortalities would be avoided when the emissions reductions from the regulation are fully realized provides additional evidence of the important role that pollution from the EGU sector plays in the public health impacts of air pollution.

Other uncertainties that we could not quantify include the importance of unquantified effects and uncertainties in the modeling of ambient air quality. Inherent in any analysis of future regulatory programs are uncertainties in projecting atmospheric conditions and source-level emissions, as well as population, health baselines, incomes, technology, and other factors. The assumptions used to capture these elements are reasonable based on the available evidence. However, data limitations prevent an overall quantitative estimate of the uncertainty associated with estimates of total economic benefits. If one is mindful of these limitations, the magnitude of the benefits estimates presented here can be useful information in expanding the understanding of the public health impacts of reducing air pollution from EGUs.

EPA will continue to evaluate new methods and models and select those most appropriate for estimating the health benefits of reductions in air pollution. It is important to continue improving benefits transfer methods in terms of transferring economic values and transferring estimated impact functions. The development of both better models of current health outcomes and new models for additional health effects such as asthma, high blood pressure, and adverse birth outcomes (such as low birth weight) will be essential to future improvements in the accuracy and reliability of benefits analyses (Guo et al., 1999; Ibald-Mulli et al., 2001). Enhanced collaboration between air quality modelers, epidemiologists, toxicologists, and economists should result in a more tightly integrated analytical framework for measuring health benefits of air pollution policies.

4.5 References

- Abbey, D.E., S.D. Colome, P.K. Mills, R. Burchette, W.L. Beeson, and Y. Tian. 1993b.
 "Chronic Disease Associated With Long-Term Concentrations of Nitrogen Dioxide." Journal of Exposure Analysis and Environmental Epidemiology 3(2):181-202.
- Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren, and P.K. Mills. 1995. "Estimated Long-Term Ambient Concentrations of PM(10) and Development of Respiratory Symptoms in a Nonsmoking Population." *Archives of Environmental Health* 50(2): 139-152.
- Abbey, D.E., N. Nishino, W.F. McDonnell, R.J. Burchette, S.F. Knutsen, W. Lawrence Beeson, and J.X. Yang. 1999. "Long-term inhalable particles and other air pollutants related to mortality in nonsmokers [see comments]." *American Journal of Respiratory and Critical Care Medicine* 159(2):373-382.
- Abt Associates, Inc. 1995. Urban Ornamental Plants: Sensitivity to Ozone and Potential Economic Losses. Prepared for the U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; Research Triangle Park, NC.
- Abt Associates, Inc. April 2003. Proposed Nonroad Land-based Diesel Engine Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results. Prepared for Office of Air Quality Planning and Standards, U.S. EPA.
- Adams, P.F., G.E. Hendershot, and M.A. Marano. 1999. "Current Estimates from the National Health Interview Survey, 1996." *Vital Health Statistics* 10(200):1-212.
- Agency for Healthcare Research and Quality (AHRQ). 2000. HCUPnet, Healthcare Cost and Utilization Project.
- American Lung Association. 1999. "Chronic Bronchitis." Available at http://www.lungusa.org/diseases/lungchronic.html.
- American Lung Association. 2002a. Trends in Asthma Morbidity and Mortality. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.
- American Lung Association. 2002b. Trends in Chronic Bronchitis and Emphysema: Morbidity and Mortality. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.

- American Lung Association. 2002c. Trends in Morbidity and Mortality: Pneumonia, Influenza, and Acute Respiratory Conditions. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.
- Ayyub, B.M. 2002. *Elicitation of Expert Opinions for Uncertainty and Risk*. CRC Press, Florida.
- Banzhaf, S., D. Burtraw, D. Evans, and A. Krupnick. September 2004. "Valuation of Natural Resource Improvements in the Adirondacks," Resources for the Future.
- Banzhaf, S., D. Burtraw, and K. Palmer. October 2002. "Efficient Emission Fees in the U.S. Electricity Sector." Resources for the Future Discussion Paper 02-45.
- Belanger, K., W. Beckett, E. Triche, M.B. Bracken, T. Holford, P. Ren, J.E. McSharry, D.R. Gold, T.A. Platts-Mills, and B.P. Leaderer. 2003. "Symptoms of Wheeze and Persistent Cough in the First Year of Life: Associations with Indoor Allergens, Air Contaminants, and Maternal History of Asthma." *American Journal of Epidemiology* 158:195-202.
- Bell, M.L., A. McDermott, S.L. Zeger, J.M. Samet, and F. Dominici. 2004. "Ozone and Short-term Mortality in 95 U.S. Urban Communities, 1987-2000." *Journal of the American Medical Association* 292:2372-2378.
- Bell, M., A. McDermott, S. Zeger, J. Samet, and F. Dominici. 2005. "A Meta-Analysis of Time-Series Studies of Ozone and Mortality with Comparison to the National Morbidity, Mortality, and Air Pollution Study." *Epidemiology* (In Press).
- Berger, M.C., G.C. Blomquist, D. Kenkel, and G.S. Tolley. 1987. "Valuing Changes in Health Risks: A Comparison of Alternative Measures." *The Southern Economic Journal* 53:977-984.
- Bricker, S.B., C.G. Clement, D.E. Pirhalla, S.P. Orlando, and D.R.G. Farrow. 1999.
 National Estuarine Eutrophication Assessment: Effects of Nutrient Enrichment in the Nation's Estuaries. National Oceanic and Atmospheric Administration, National Ocean Service, Special Projects Office and the National Centers for Coastal Ocean Science. Silver Spring, Maryland.

- Burnett, R.T., M. Smith-Doiron, D. Stieb, M.E. Raizenne, J.R. Brook, R.E Dales, J.A. Leech, S. Cakmak, D. Krewski. 2001. "Association between Ozone and Hospitalization for Acute Respiratory Diseases in Children less than 2 Years of Age." *American Journal* of Epidemiology 153:444-52.
- CDC Wonder, Wide-ranging OnLine Data for Epidemiologic Research (Wonder) (data from years 1996-1998), Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, http://wonder.cdc.gov.
- Carnethon, M.R., D. Liao, G.W. Evans, W.E. Cascio, L.E. Chambless, W.D. Rosamond, and G. Heiss. 2002. "Does the Cardiac Autonomic Response to Postural Change Predict Incident Coronary Heart Disease and Mortality? The Atherosclerosis Risk in Communities Study." *American Journal of Epidemiology* 155(1):48-56.
- Chay, K.Y., and M. Greenstone. 2003. "The Impact of Air Pollution on Infant Mortality: Evidence from Geographic Variation in Pollution Shocks Induced by a Recession." *Quarterly Journal of Economics* 118(3).
- Chen, L., B.L. Jennison, W. Yang, and S.T. Omaye. 2000. "Elementary School Absenteeism and Air Pollution." *Inhalation Toxicology* 12(11):997-1016.
- Chestnut, L.G. April 15, 1997. Draft Memorandum: Methodology for Estimating Values for Changes in Visibility at National Parks.
- Chestnut, L.G. and R.L. Dennis. 1997. "Economic Benefits of Improvements in Visibility: Acid Rain Provisions of the 1990 Clean Air Act Amendments." *Journal of the Air and Waste Management Association* 47:395-402.
- Chestnut, L.G., and R.D. Rowe. 1990a. "A New National Park Visibility Value Estimates." In Visibility and Fine Particles, Transactions of an AWMA/EPA International Specialty Conference, C.V. Mathai, ed. Air and Waste Management Association, Pittsburgh.
- Chestnut, L.G., and R.D. Rowe. 1990b. Preservation Values for Visibility Protection at the National Parks: Draft Final Report. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC, and Air Quality Management Division, National Park Service, Denver, CO.

- Cody, R.P., C.P. Weisel, G. Birnbaum, and P.J. Lioy. 1992. "The Effect of Ozone Associated with Summertime Photochemical Smog on the Frequency of Asthma Visits to Hospital Emergency Departments." *Environmental Research* 58(2):184-94.
- Crocker, T.D., and R.L. Horst, Jr. 1981. "Hours of Work, Labor Productivity, and Environmental Conditions: A Case Study." *The Review of Economics and Statistics* 63:361-368.
- Cropper, M.L., and A.J. Krupnick. 1990. "The Social Costs of Chronic Heart and Lung Disease." Resources for the Future. Washington, DC. Discussion Paper QE 89-16-REV.
- Daniels, M.J., F. Dominici, J.M. Samet, and S.L. Zeger. 2000. "Estimating Particulate Matter-Mortality Dose-Response Curves and Threshold Levels: An Analysis of Daily Time-Series for the 20 Largest U.S. Cities." *American Journal of Epidemiology* 152(5):397-406.
- Dekker, J.M., R.S. Crow, A.R. Folsom, P.J. Hannan, D. Liao, C.A. Swenne, and E.G. Schouten. 2000. "Low Heart Rate Variability in a 2-Minute Rhythm Strip Predicts Risk of Coronary Heart Disease and Mortality From Several Causes: The ARIC Study." *Circulation 2000* 102:1239-1244.
- Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris, and F.E. Speizer. 1993. "An Association between Air Pollution and Mortality in Six U.S. Cities." *New England Journal of Medicine* 329(24):1753-1759.
- Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne, and F.E. Speizer. 1996. "Health Effects of Acid Aerosols On North American Children-Respiratory Symptoms." *Environmental Health Perspectives* 104(5):500-505.
- Eisenstein, E.L., L.K. Shaw, K.J. Anstrom, C.L. Nelson, Z. Hakim, V. Hasselblad and D.B. Mark. 2001. "Assessing the Clinical and Economic Burden of Coronary Artery Disease: 1986-1998." *Medical Care* 39(8):824-35.
- EPA-SAB-COUNCIL-ADV-98-003. 1998. "Advisory Council on Clean Air Compliance Analysis Advisory on the Clean Air Act Amendments (CAAA) of 1990 Section 812 Prospective Study: Overview of Air Quality and Emissions Estimates: Modeling, Health and Ecological Valuation Issues Initial Studies."

- EPA-SAB-COUNCIL-ADV-99-012. July 1999. "The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects: Part 1."
- EPA-SAB-COUNCIL-ADV-99-005. February 1999. "An SAB Advisory on the Health and Ecological Effects Initial Studies of the Section 812 Prospective Study: Report to Congress: Advisory by the Health and Ecological Effects Subcommittee."
- EPA-SAB-COUNCIL-ADV-01-004. 2001. "Review of the Draft Analytical Plan for EPA's Second Prospective Analysis—Benefits and Costs of the Clean Air Act 1990-2020: An Advisory by a Special Panel of the Advisory Council on Clean Air Compliance Analysis." September.
- EPA-SAB-EEAC-00-013. July 2000. "An SAB Report on EPA's White Paper Valuing the Benefits of Fatal Cancer Risk Reduction."
- Fox, S., and R.A. Mickler. 1996. "Impact of Air Pollutants on Southern Pine Forests." *Ecological Studies* 118. New York: Springer Verlag.
- Freeman, A.M. III. 1993. *The Measurement of Environmental and Resource Values: Theory and Methods*. Washington, DC: Resources for the Future.
- Garcia, P., B. Dixon, and J. Mjelde. 1986. "Measuring the Benefits of Environmental Change Using a Duality Approach: The Case of Ozone and Illinois Cash Grain Farms." *Journal of Environmental Economics and Management* 13:69-80.
- Gilliland, F.D., K. Berhane, E.B. Rappaport, D.C. Thomas, E. Avol, W.J. Gauderman, S.J. London, H.G. Margolis, R. McConnell, K.T. Islam, and J.M. Peters. 2001. "The Effects of Ambient Air Pollution on School Absenteeism due to Respiratory Illnesses." *Epidemiology* 12(1):43-54.
- Gold, D.R., A. Litonjua, J. Schwartz, E. Lovett, A. Larson, B. Nearing, G. Allen, M. Verrier, R. Cherry., and R. Verrier. 2000. "Ambient Pollution and Heart Rate Variability." *Circulation* 101(11):1267-73.

- Greenbaum, D. 2002. Letter to colleagues dated May 30, 2002. [Available at www.healtheffects.org]. Letter from L.D. Grant, Ph.D. to Dr. P. Hopke re: external review of EPA's Air Quality Criteria for Particulate Matter, with copy of 05/30/02 letter from Health Effects Institute re: re-analysis of National Morbidity, Mortality and Air Pollution Study data attached. Docket No. A-2000-01. Document No. IV-A-145.
- Grosclaude, P., and N.C. Soguel. 1994. "Valuing Damage to Historic Buildings Using a Contingent Market: A Case Study of Road Traffic Externalities." *Journal of Environmental Planning and Management* 37: 279-287.
- Guo, Y.L., Y.C. Lin, F.C. Sung, S.L. Huang, Y.C. Ko, J.S. Lai, H.J. Su, C.K. Shaw, R.S. Lin, and D.W. Dockery. 1999. "Climate, Traffic-Related Air Pollutants, and Asthma Prevalence in Middle-School Children in Taiwan." *Environmental Health Perspectives* 107:1001-1006.
- Hall, J.V., V. Brajer, and F.W. Lurmann. 2003. "Economic Valuation of Ozone-related School Absences in the South Coast Air Basin of California." *Contemporary Economic Policy* 21(4):407-417.
- Harrington, W., and P.R. Portney. 1987. "Valuing the Benefits of Health and Safety Regulation." *Journal of Urban Economics* 22:101-112.
- Health Effects Institute (HEI). 2003. Revised Analyses of Time-Series Studies of Air Pollution and Health; Revised Analyses of the National Morbidity, Mortality and Air Pollution Study, Part II; Revised Analyses of Selected Time-Series Studies. Health Effects Institute, Boston, MA.
- Hollman, F.W., T.J. Mulder, and J.E. Kallan. January 2000. "Methodology and Assumptions for the Population Projections of the United States: 1999 to 2100." Population Division Working Paper No. 38, Population Projections Branch, Population Division, U.S. Census Bureau, Department of Commerce.
- Ibald-Mulli, A., J. Stieber, H.-E. Wichmann, W. Koenig, and A. Peters. 2001. "Effects of Air Pollution on Blood Pressure: A Population-Based Approach." *American Journal* of Public Health 91:571-577.
- Industrial Economics, Incorporated (IEc). March 31., 1994. Memorandum to Jim DeMocker, Office of Air and Radiation, Office of Policy Analysis and Review, U.S. Environmental Protection Agency.

- Industrial Economics, Incorporated (IEc). April 2004. "Expert Judgment Assessment of the Relationship Between PM_{2.5} Exposure and Mortality." Available at www.epa.gov/ttn/ecas/benefits.html.
- Ito, K. 2003. "Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit, Michigan." In *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Special Report. Health Effects Institute, Boston, MA.
- Ito, K., S. De Leon, and M. Lippman. 2005. Associations between ozone and daily mortality: a review and an additional analysis. Epidemiology (In Press).
- Ito, K., and G.D. Thurston. 1996. "Daily PM10/Mortality Associations: An Investigations of At-Risk Subpopulations." *Journal of Exposure Analysis and Environmental Epidemiology* 6(1):79-95.
- Ito, K., and G.D. Thurston. 2001. "Epidemiological studies of acute ozone exposures and mortality." *Journal of Exposure Analysis and Environmental Epidemiology* 11(4):286-294.
- Kleckner, N., and J. Neumann. June 3, 1999. "Recommended Approach to Adjusting WTP Estimates to Reflect Changes in Real Income." Memorandum to Jim Democker, U.S. EPA/OPAR.
- Krewski D., R.T. Burnett, M.S. Goldbert, K. Hoover, J. Siemiatycki, M. Jerrett, M.
 Abrahamowicz, and W.H. White. July 2000. *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality*. Special Report to the Health Effects Institute, Cambridge MA.
- Krupnick, A.J., and M.L. Cropper. 1992. "The Effect of Information on Health Risk Valuations." *Journal of Risk and Uncertainty* 5(2):29-48.
- Kunzli, N., S. Medina, R. Kaiser, P. Quenel, F. Horak Jr, and M. Studnicka. 2001.
 "Assessment of Deaths Attributable to Air Pollution: Should We Use Risk Estimates Based on Time Series or on Cohort Studies?" *American Journal of Epidemiology* 153(11):1050-55.
- Kunzli, N., R. Kaiser, S. Medina, M. Studnicka, O. Chanel, P. Filliger, M. Herry, F. Horak Jr., V. Puybonnieux-Texier, P. Quenel, J. Schneider, R. Seethaler, J-C Vergnaud, and H. Sommer. 2000. "Public-Health Impact of Outdoor and Traffic-Related Air Pollution: A European Assessment." *The Lancet* 356:795-801.

- Lave, L.B., and E.P. Seskin. 1977. *Air Pollution and Human Health*. Baltimore: Johns Hopkins University Press for Resources for the Future.
- Levy, J.I., J.K. Hammitt, Y. Yanagisawa, and J.D. Spengler. 1999. "Development of a New Damage Function Model for Power Plants: Methodology and Applications." *Environmental Science and Technology* 33:4364-4372.
- Levy, J.I., T.J. Carrothers, J.T. Tuomisto, J.K. Hammitt, and J.S. Evans. 2001. "Assessing the Public Health Benefits of Reduced Ozone Concentrations." *Environmental Health Perspectives* 109:1215-1226.
- Levy, J.I., S. M. Chemerynski, and J.A. Sarnat. 2005. "Ozone Exposure and Mortality Risk: An Empirical Bayes Meta-Regression Analysis." *Epidemiology* (In Press).
- Liao, D., J. Cai, W.D. Rosamond, R.W. Barnes, R.G. Hutchinson, E.A. Whitsel, P. Rautaharju, and G. Heiss. 1997. "Cardiac Autonomic Function and Incident Coronary Heart Disease: A Population-Based Case-Cohort Study. The ARIC Study. Atherosclerosis Risk in Communities Study." *American Journal of Epidemiology* 145(8):696-706.
- Liao, D., J. Creason, C. Shy, R. Williams, R. Watts, and R. Zweidinger. 1999. "Daily Variation of Particulate Air Pollution and Poor Cardiac Autonomic Control in the Elderly." *Environ Health Perspect* 107:521-5.
- Lipfert, F.W., S.C. Morris, and R.E. Wyzga. 1989. "Acid Aerosols—the Next Criteria Air Pollutant." *Environmental Science & Technology* 23(11):1316-1322.
- Lipfert, F.W., H. Mitchell Perry Jr., J. Philip Miller, Jack D. Baty, Ronald E. Wyzg, and Sharon E. Carmody. 2000. "The Washington University-EPRI Veterans' Cohort Mortality Study: Preliminary Results." *Inhalation Toxicology* 12:41-74.
- Magari, S.R., R. Hauser, J. Schwartz, P.L. Williams, T.J. Smith, and D.C. Christiani. 2001.
 "Association of Heart rate Variability with Occupational and Environmental Exposure to Particulate Air Pollution." *Circulation* 104(9):986-91.
- Mansfield, C. 2004. "Peer Review of Expert Elicitation." Research Triangle Park, NC. RTI International. Available at: </www.epa.gov/ttn/ecase/benefits.html>.

- McClelland, G., W. Schulze, D. Waldman, J. Irwin, D. Schenk, T. Stewart, L. Deck, and M. Thayer. September 1993. *Valuing Eastern Visibility: A Field Test of the Contingent Valuation Method*. Prepared for Office of Policy, Planning and Evaluation, U.S. Environmental Protection Agency.
- McConnell, R., K. Berhane, F. Gilliland, S.J. London, H. Vora, E. Avol, W.J. Gauderman, H.G. Margolis, F. Lurmann, D.C. Thomas, and J.M. Peters. 1999. "Air Pollution and Bronchitic Symptoms in Southern California Children with Asthma." *Environmental Health Perspectives* 107(9):757-760.
- McConnell, R., K. Berhane, F. Gilliland, S.J. London, T. Islam, W.J. Gauderman, E. Avol, H.G. Margolis, and J.M. Peters. 2002. "Asthma in Exercising Children Exposed to Ozone: A Cohort Study." *Lancet* 359(9309):896.
- Moolgavkar, S.H. 2000. "Air Pollution and Hospital Admissions for Diseases of the Circulatory System in Three U.S. Metropolitan Areas." *Journal of the Air and Waste Management Association* 50:1199-1206.
- Moolgavkar, S.H. 2003. "Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties." In *Revised Analyses of Time-Series Studies of Air Pollution and Health.* Special Report. Boston, MA: Health Effects Institute.
- Moolgavkar, S.H., E.G. Luebeck, and E.L. Anderson. 1997. "Air Pollution and Hospital Admissions for Respiratory Causes in Minneapolis-St. Paul and Birmingham." *Epidemiology* 8:364-370.
- Mrozek, J.R., and L.O. Taylor. 2002. "What Determines the Value of Life? A Meta-Analysis." *Journal of Policy Analysis and Management* 21(2):253-270.
- National Hospital Ambulatory Medical Care Survey (NHAMCS). 2000. National Center for Health Statistics, Centers for Disease Control and Prevention (CDC), US Department of Health and Human Services. Available at: http://www.cdc.gov/nchs/about/major/ ahcd/ahcd1.htm>.
- NHDS Public Use Data Files. 1999. Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services. Available at: ftp://ftp.cdc.gov/pub/ Health_Statistics/NCHS/datasets/NHDS.

- National Health Interview Survey (NHIS). 2000. National Center for Health Statistics, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Web site: ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/ Datasets/NHIS/2000/.
- National Center for Education Statistics. 1996. "The Condition of Education 1996, Indicator 42: Student Absenteeism and Tardiness." Washington, DC: U.S. Department of Education National Center for Education Statistics.
- National Research Council (NRC). 1998. Research Priorities for Airborne Particulate Matter: I. Immediate Priorities and a Long-Range Research Portfolio. Washington, DC: The National Academies Press.
- National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.
- Neumann, J.E., M.T. Dickie, and R.E. Unsworth. March 31, 1994. "Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis—Draft Valuation Document." Industrial Economics Incorporated (IEc) Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review.
- Norris, G., S.N. YoungPong, J.Q. Koenig, T.V. Larson, L. Sheppard, and J.W. Stout. 1999.
 "An Association between Fine Particles and Asthma Emergency Department Visits for Children in Seattle." *Environmental Health Perspectives* 107(6):489-493.
- Ostro, B.D. 1987. "Air Pollution and Morbidity Revisited: A Specification Test." *Journal of Environmental Economics Management* 14:87-98.
- Ostro, B.D. and S. Rothschild. 1989. "Air Pollution and Acute Respiratory Morbidity: An Observational Study of Multiple Pollutants." *Environmental Research* 50:238-247.
- Ostro, B.D., M.J. Lipsett, M.B. Wiener, and J.C. Selner. 1991. "Asthmatic Responses to Airborne Acid Aerosols." *American Journal of Public Health* 81(6):694-702.
- Ostro, B., and L. Chestnut. 1998. "Assessing the Health Benefits of Reducing Particulate Matter Air Pollution in the United States." Environmental Research, Section A, 76:94-106.

- Ostro, B., M. Lipsett, J. Mann, H. Braxton-Owens, and M. White. 2001. "Air Pollution and Exacerbation of Asthma in African-American Children in Los Angeles." *Epidemiology* 12(2):200-208.
- Ozkaynak, H., and G.D. Thurston. 1987. "Associations between 1980 U.S. Mortality Rates and Alternative Measures of Airborne Particle Concentration." *Risk Analysis* 7(4):449-461.
- Peters, A., D.W. Dockery, J.E. Muller, and M.A. Mittleman. 2001. "Increased Particulate Air Pollution and the Triggering of Myocardial Infarction." *Circulation* 103:2810-2815.
- Poloniecki, J.D., R.W. Atkinson., A.P. de Leon., and H.R. Anderson. 1997. "Daily Time Series for Cardiovascular Hospital Admissions and Previous Day's Air Pollution in London, UK." *Occupational and Environmental Medicine* 54(8):535-540.
- Pope, C.A. 2000. "Invited Commentary: Particulate Matter-Mortality Exposure-Response Relations and Thresholds." *American Journal of Epidemiology* 152:407-412.
- Pope, C.A., III, M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer, and C.W. Heath, Jr. 1995. "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults." *American Journal of Respiratory Critical Care Medicine* 151:669-674.
- Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- Pope, C.A., III, D.W. Dockery, J.D. Spengler, and M.E. Raizenne. 1991. "Respiratory Health and PM₁₀ Pollution: A Daily Time Series Analysis." *American Review of Respiratory Diseases* 144:668-674.
- Ransom, M.R., and C.A. Pope. 1992. "Elementary School Absences and PM(10) Pollution in Utah Valley." *Environmental Research* 58(2):204-219.
- Rosamond, W., G. Broda, E. Kawalec, S. Rywik, A. Pajak, L. Cooper, and L. Chambless.
 1999. "Comparison of Medical Care and Survival of Hospitalized Patients with Acute Myocardial Infarction in Poland and the United States." *American Journal of Cardiology* 83:1180-1185.

- Rossi, G., M.A. Vigotti, A. Zanobetti, F. Repetto, V. Gianelle, and J. Schwartz. 1999. "Air Pollution and Cause-Specific Mortality in Milan, Italy, 1980-1989." Archives of Environmental Health 54(3):158-164
- Rowe, R.D., and L.G. Chestnut. 1986. "Oxidants and Asthmatics in Los Angeles: A Benefits Analysis—Executive Summary." Prepared by Energy and Resource Consultants, Inc. Report to the U.S. Environmental Protection Agency, Office of Policy Analysis. EPA-230-09-86-018. Washington, DC.
- Rowlatt, et al. 1998. "Valuation of Deaths from Air Pollution." NERA and CASPAR for DETR.
- Russell, M.W., D.M. Huse, S. Drowns, E.C. Hamel, and S.C. Hartz. 1998. "Direct Medical Costs of Coronary Artery Disease in the United States." *American Journal of Cardiology* 81(9):1110-1115.
- Samet, J.M., S.L. Zeger, J.E. Kelsall, J. Xu, and L.S. Kalkstein. March 1997. "Air Pollution, Weather, and Mortality in Philadelphia 1973-1988." Cambridge, MA: Health Effects Institute.
- Samet, J.M., S.L. Zeger, F. Dominici, F. Curriero, I. Coursac, D.W. Dockery, J. Schwartz, and A. Zanobetti. June 2000. *The National Morbidity, Mortality and Air Pollution Study: Part II: Morbidity, Mortality and Air Pollution in the United States*. Research Report No. 94, Part II. Health Effects Institute, Cambridge MA.
- Schwartz, J. 1993. "Particulate Air Pollution and Chronic Respiratory Disease." *Environmental Research* 62:7-13.
- Schwartz, J. 1994a. "PM(10) Ozone, and Hospital Admissions for the Elderly in Minneapolis-St Paul, Minnesota." Archives of Environmental Health 49(5):366-374.
- Schwartz, J. 1994b. "Air Pollution and Hospital Admissions for the Elderly in Detroit, Michigan." American Journal of Respiratory and Critical Care Medicine 150(3):648-655.
- Schwartz, J. 1995. "Short Term Fluctuations in Air Pollution and Hospital Admissions of the Elderly for Respiratory Disease." *Thorax* 50(5):531-538

- Schwartz, J., D.W. Dockery, L.M. Neas, D. Wypij, J.H. Ware, J.D. Spengler, P. Koutrakis, F.E. Speizer, and B.G. Ferris, Jr. 1994. "Acute Effects of Summer Air Pollution on Respiratory Symptom Reporting in Children." *American Journal of Respiratory Critical Care Medicine* 150:1234-1242.
- Schwartz, J., and A. Zanobetti. 2000. "Using Meta-Smoothing to Estimate Dose-Response Trends across Multiple Studies, with Application to Air Pollution and Daily Death." *Epidemiology* 11:666-672.
- Schwartz, J., and L.M. Neas. 2000. "Fine Particles are More Strongly Associated than Coarse Particles with Acute Respiratory Health Effects in Schoolchildren." *Epidemiology* 11:6-10.
- Schwartz, J., F. Laden, and A. Zanobetti. 2002. "The Concentration-Response Relation between PM(2.5) and Daily Deaths." *Environmental Health Perspectives* 110:1025-1029.
- Sheppard, L. 2003. "Ambient Air Pollution and Nonelderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994." In *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Special Report. Boston, MA: Health Effects Institute.
- Sheppard, L., D. Levy, G. Norris, T.V. Larson, and J.Q. Koenig. 1999. "Effects of Ambient Air Pollution on Nonelderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994." *Epidemiology* 10:23-30.
- Shogren, J., and T. Stamland. 2002. "Skill and the Value of Life." *Journal of Political Economy* 110:1168-1197.
- Sisler, J.F. 1996. Spatial and Seasonal Patterns and Long Term Variability of the Composition of the Haze in the United States: An Analysis of Data from the IMPROVE Network. Colorado State University, Cooperative Institute for Research in the Atmosphere. Fort Collins, CO. July. See EPA Air Docket A-96-56, Document No. VI-B-09-(ee).
- Smith, A., T. Kim, M. Fuentes, and D. Spitzner. 2000. "Threshold Dependence of Mortality Effects for Fine and Coarse Particles in Phoenix, Arizona." *Journal of the Air and Waste Management Association* 5:1367-1379.

- Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista, and W.B. Saunders. 1997. "A National Estimate of the Economic Costs of Asthma." *American Journal of Respiratory and Critical Care Medicine* 156(3 Pt 1):787-793.
- Smith, V.K., G. Van Houtven, and S.K. Pattanayak. 2002. "Benefit Transfer via Preference Calibration." *Land Economics* 78:132-152.
- Standard and Poor's. 2000. "The U.S. Economy: The 25 Year Focus." Winter. Web site: http://www2standardandpoors.com.
- Stanford, R., T. McLaughlin, and L.J. Okamoto. 1999. "The Cost of Asthma in the Emergency Department and Hospital." *American Journal of Respiratory and Critical Care Medicine* 160(1):211-215.
- Stieb, D.M., R.T. Burnett, R.C. Beveridge, and J.R. Brook. 1996. "Association between Ozone and Asthma Emergency Department Visits in Saint John, New Brunswick, Canada." *Environmental Health Perspectives* 104(12):1354-1360.
- Sweeney, Jeff. "EPA's Chesapeake Bay Program Air Strategy." October 26, 2004.
- Thurston, G.D., and K. Ito. 2001. "Epidemiological Studies of Acute Ozone Exposures and Mortality." *Journal of Exposure Analysis and Environmental Epidemiology* 11(4):286-294.
- Tolley, G.S. et al. January 1986. *Valuation of Reductions in Human Health Symptoms and Risks. University of Chicago*. Final Report for the U.S. Environmental Protection Agency.
- Tsuji, H., M.G. Larson, F.J. Venditti, Jr., E.S. Manders, J.C. Evans, C.L. Feldman, D. Levy. 1996. "Impact of Reduced Heart Rate Variability on Risk for Cardiac Events. The Framingham Heart Study." *Circulation* 94(11):2850-2855.
- U.S. Bureau of Census. Annual Projections of the Total Resident Population, Middle Series, 1999–2100. Available at: < http://www.census.gov/population/www/projections/natsum-T1.html>.
- U.S. Bureau of Census. 2000. Population Projections of the United States by Age, Sex, Race, Hispanic Origin and Nativity: 1999 to 2100. Population Projections Program, Population Division, U.S. Census Bureau, Washington, DC. Available at: http://www.census.gov/population/projections/nation/summary/np-t.txt.

- U.S. Bureau of the Census. 2002. *Statistical Abstract of the United States: 2001.* Washington, DC.
- Floriculture and Nursery Crops Situation and Outlook Yearbook. 2004. Market and Trade Economics Division, Economic Research Service, U.S. Department of Agriculture, FLO-2004.
- NPS/NERCHAL/NRTR-03/090. Philadelphia, PA: National Park Service, Northeast Region. Available at http://www.nps.gov/shen/air_quality.htm.
- U.S. Environmental Protection Agency (EPA). 1993. External Draft, Air Quality Criteria for Ozone and Related Photochemical Oxidants. Volume II. U.S. EPA, Office of Health and Environmental Assessment. Research Triangle Park, NC, EPA/600/AP-93/004b.3v.
- U.S. Environmental Protection Agency (EPA). 1996a. Review of the National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information. Office of Air Quality Planning and Standards, Research Triangle Park, NC, EPA report no. EPA/4521R-96-007.
- U.S. Environmental Protection Agency (EPA). 1996b. Review of the National Ambient Air Quality Standards for Particulate Matter: Assessment of Scientific and Technical Information. Office of Air Quality Planning and Standards, Research Triangle Park, NC; EPA report no. EPA/4521R-96-013.
- U.S. Environmental Protection Agency (EPA). 1997. *The Benefits and Costs of the Clean Air Act, 1970 to 1990.* Prepared for U.S. Congress by U.S. EPA, Office of Air and Radiation/Office of Policy Analysis and Review, Washington, DC.
- U.S. Environmental Protection Agency (EPA). 1999a. The Benefits and Costs of the Clean Air Act, 1990-2010. Prepared for U.S. Congress by U.S. EPA, Office of Air and Radiation/Office of Policy Analysis and Review, Washington, DC, November; EPA report no. EPA-410-R-99-001.
- U.S. Environmental Protection Agency (EPA). 1999b. "An SAB Advisory: The Clean Air Act Section 812 Prospective Study Health and Ecological Initial Studies." Prepared by the Health and Ecological Effects Subcommittee (HEES) of the Advisory Council on the Clean Air Compliance Analysis, Science Advisory Board, U.S. Environmental Protection Agency. Washington DC. EPA-SAB-COUNCIL-ADV-99-005.

- U.S. Environmental Protection Agency (EPA). September 2000b. *Guidelines for Preparing Economic Analyses*. EPA 240-R-00-003.
- U.S. Environmental Protection Agency (EPA). 2000c. Integrated Risk Information System; website access available at www.epa.gov/ngispgm3/iris. Data as of December 2000.
- U.S. Environmental Protection Agency (EPA). 2000d. *Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements.* Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/otaq/diesel.htm. Accessed March 20, 2003.
- U.S. Environmental Protection Agency. 2003c. Clear Skies Act—Technical Report: Section B.
- U.S. Environmental Protection Agency. January 2004a. Air Quality Data Analysis Technical Support Document for the Proposed Interstate Air Quality Rule.
- U.S. Nuclear Regulatory Commission (NRC). 1996. "Branch Technical Position on the Use of Expert Elicitation in the High-Level Radioactive Waste Program." November, 1996.
- U.S. Office of Management and Budget. October 1992. "Guidelines and Discount Rates for Benefit-Cost Analysis of Federal Programs." Circular No. A-94.
- U.S. Office of Management and Budget (OMB). 2003. Circular A-4 Guidance for Federal Agencies Preparing Regulatory Analyses, Available at: http://www/whitehouse.gov/omb/inforeg/iraguide.html.
- Vedal, S., J. Petkau, R. White, and J. Blair. 1998. "Acute Effects of Ambient Inhalable Particles in Asthmatic and Nonasthmatic Children." *American Journal of Respiratory* and Critical Care Medicine 157(4):1034-1043.
- Viscusi, W.K., W.A. Magat, and J. Huber. 1991. "Pricing Environmental Health Risks: Survey Assessments of Risk-Risk and Risk-Dollar Trade-Offs for Chronic Bronchitis." *Journal of Environmental Economics and Management* 21:32-51.
- Viscusi, V.K., and J.E. Aldy. 2003. "The Value of a Statistical Life: A Critical Review of Market Estimates Throughout the World." *Journal of Risk and Uncertainty* 27(1):5-76.

- Weisel, C.P., R.P. Cody, and P.J. Lioy. 1995. "Relationship between Summertime Ambient Ozone Levels and Emergency Department Visits for Asthma in Central New Jersey." *Environmental Health Perspectives* 103 Suppl 2:97-102.
- Whittemore, A.S., and E.L. Korn. 1980. "Asthma and Air Pollution in the Los Angeles Area." *American Journal of Public Health* 70:687-696.
- Wittels, E.H., J.W. Hay, and A.M. Gotto, Jr. 1990. "Medical Costs of Coronary Artery Disease in the United States." *American Journal of Cardiology* 65(7):432-440.
- Woodruff, T.J., J. Grillo, and K.C. Schoendorf. 1997. "The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Air Pollution in the United States." *Environmental Health Perspectives* 105(6):608-612.
- Woods & Poole Economics, Inc. 2001. "Population by Single Year of Age CD." Woods & Poole Economics, Inc.
- World Health Organization (WHO). 2002. "Global Burden of Disease Study." World Health Organization.
- Yu, O., L. Sheppard, T. Lumley, J.Q. Koenig, and G.G. Shapiro. 2000. "Effects of Ambient Air Pollution on Symptoms of Asthma in Seattle-Area Children Enrolled in the CAMP Study." *Environmental Health Perspect*ives 108(12):1209-1214.

CHAPTER 5

QUALITATIVE ASSESSMENT OF NONMONETIZED BENEFITS

5.1 Introduction

In addition to the enumerated human health and welfare benefits resulting from reductions in ambient levels of PM and ozone, CAIR will result in benefits that we are not able to monetize. This chapter discusses welfare benefits associated with reduced sulfur and nitrogen deposition that affects acidification of ecosystems and eutrophication in water bodies. Other welfare benefits including potential visibility improvements, agricultural yield increases, forestry production increases, reductions in soiling and materials damage, mercury health and welfare benefits, and other welfare categories are discussed in Chapter 4 of this report.

5.2 Atmospheric Deposition of Sulfur and Nitrogen—Quantification of Impacts for the Rule

The benefits associated with reduced sulfur and nitrogen deposition to aquatic, forest, and coastal ecosystems are qualitatively discussed in this section. We also quantify the reductions in acid deposition for the nation's lakes in the northeast, including the Adirondacks, and in southeast streams that are expected to occur as a result of this rule. Figures 5-1 and 5-2 present the reductions in sulfur and nitrogen deposition anticipated to occur in 2010 and 2015 in the Eastern United States as a result of emission reductions anticipated under the final CAIR. As shown in Figure 5-1, CAIR is predicted to result in sulfur deposition reductions ranging from slightly less than 1 to 59 percent in specific areas of the CAIR region and other eastern states with reductions of 16.6 percent on average for the areas east of the 100th parallel in 2015. As Figure 5-2 depicts, changes in all forms of nitrogen deposition in the region are expected to range from small areas of slight increases that do not exceed 1.5 percent to reductions up to 19 percent and average reductions of 4.6 percent for areas east of the 100th parallel in 2015.

Figure 5-3 reports average nitrogen and sulfur deposition reductions anticipated for the Adirondacks, New England, the Blue Ridge in 2015 under CAIR. As shown in Figure 5-3, sulfur depositions reductions from baseline conditions are predicted in 2015 of

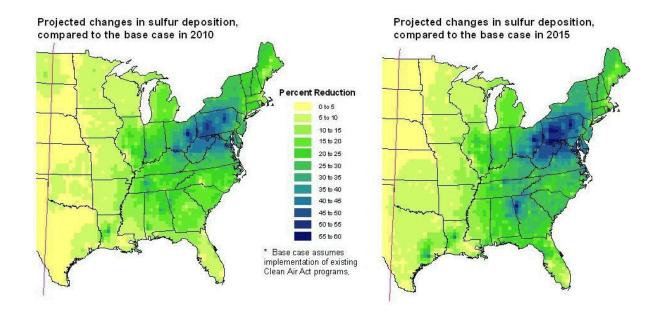


Figure 5-1. Percentage Reduction of All Forms of Sulfur Deposition for the Years 2010 and 2015

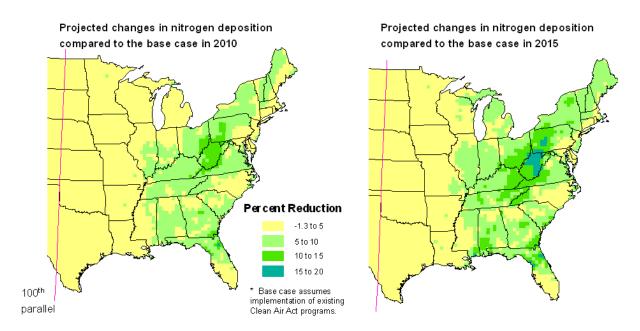


Figure 5-2. Percentage Reduction of All Forms of Nitrogen Deposition for the Years 2010 and 2015

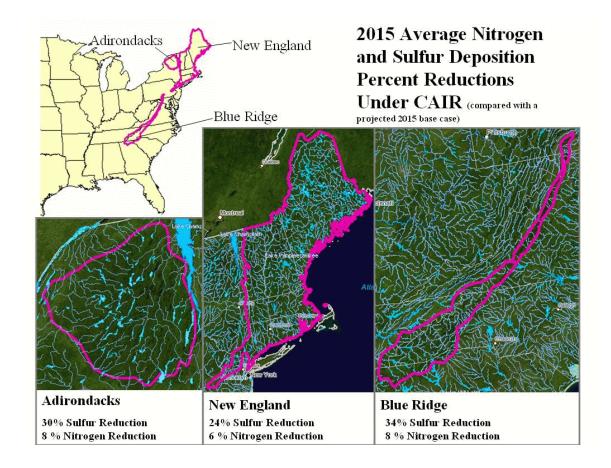


Figure 5-3. CAIR Nitrogen and Sulfur Deposition Reductions in the Adirondacks, New England, and the Blue Ridge

approximately 30 percent in the Adirondacks, 24 percent in New England, and 34 percent in the Blue Ridge. In 2015, these same regions are expected to experience nitrogen deposition reductions ranging from 6 to 8 percent from baseline conditions.

5.3 Atmospheric Deposition of Sulfur and Nitrogen—Impacts on Aquatic, Forest, and Coastal Ecosystems

Atmospheric deposition of sulfur and nitrogen, more commonly known as acid rain, occurs when emissions of SO_2 and NO_x react in the atmosphere (with water, oxygen, and oxidants) to form various acidic compounds. These acidic compounds fall to Earth in either a wet form (rain, snow, and fog) or a dry form (gases and particles). Prevailing winds transport the acidic compounds hundreds of miles, often across state and national borders. Acidic compounds (including small particles such as sulfates and nitrates) cause many negative environmental effects. These pollutants

- acidify lakes and streams,
- harm sensitive forests, and
- harm sensitive coastal ecosystems.

The effect of atmospheric deposition of acids on freshwater and forest ecosystems depends largely on the ecosystem's ability to neutralize the acid (Driscoll et al., 2001). This is referred to as an ecosystem's acid neutralizing capacity (ANC). Acid neutralization occurs when positively charged ions such as calcium, potassium, sodium, and magnesium, collectively known as base cations, are released. As water moves through a watershed, two important chemical processes act to neutralize acids. The first involves cation exchange in soils, a process by which hydrogen ions from the acid deposition displace other cations from the surface of soil particles, releasing these cations to soil and surface water. The second process is mineral weathering, where base cations bound in the mineral structure of rocks are released as the minerals gradually break down over long time periods. As the base cations are released by weathering, they neutralize acidity and increase the pH level in soil water and surface waters. Acid deposition, because it consists of acid anions (e.g., sulfate, nitrate), leaches some of the accumulated base cation reserves from the soils into drainage waters. The leaching rate of these base cations may accelerate to the point where it significantly exceeds the resupply via weathering (Driscoll et al., 2001). CAIR is expected to reduce atmospheric deposition of nitrogen and sulfur and to reduce the total nitrogen and sulfur load in areas of the East.

Soils, forests, surface waters and aquatic biota (fish, algae, and the rest), and coastal ecosystems share water, nutrients, and other essential ecosystem components and are inextricably linked by the chemical processes described above. For example, the same base cations that help to neutralize acidity in lakes and streams are also essential nutrients in forest soils, meaning that cation depletion both increases freshwater acidification and decreases forest productivity. Similarly, the same nitrogen atom that contributes to stream acidification can ultimately contribute to coastal eutrophication as it travels downstream to an estuarine environment. Therefore, to understand the full effects of atmospheric deposition, it is necessary to recognize the interactions between all of these systems.

5.3.1 Freshwater Acidification

Acid deposition causes acidification of surface waters. In the 1980s, acid rain was found to be the dominant cause of acidification in 75 percent of acidic lakes and 50 percent of acidic streams. Areas especially sensitive to acidification include portions of the

Northeast (particularly the Adirondack and Catskill Mountains, portions of New England, and streams in the mid-Appalachian highlands) and Southeastern streams. Some highelevation Western lakes, particularly in the Rocky Mountains, have become acidic, especially during snowmelt. However, although many Western lakes and streams are sensitive to acidification, they are not subject to continuously high levels of acid deposition and so have not become chronically acidified (NAPAP, 1990).

ANC, a key indicator of the ability of the water and watershed soil to neutralize the acid deposition it receives, depends largely on the watershed's physical characteristics: geology, soils, and size. Waters that are sensitive to acidification tend to be located in small watersheds that have few alkaline minerals and shallow soils. Conversely, watersheds that contain alkaline minerals, such as limestone, tend to have waters with a high ANC.

As acidity increases, aluminum leached from the soil flows into lakes and streams and can be toxic to aquatic species. The lower pH levels and higher aluminum levels that result from acidification make it difficult for some fish and other aquatic species to survive, grow, and reproduce. In some waters, the number of species of fish able to survive has been directly correlated to water acidity. Acidification can also decrease fish population density and individual fish size (U.S. Department of the Interior, 2003).

Recent watershed mass balance studies in the Northeast reveal that loss of sulfate from the watershed exceeds atmospheric sulfur deposition (Driscoll et al., 2001). This suggests that these soils have become saturated with sulfur, meaning that the supply of sulfur from deposition exceeds the sulfur demands of the ecosystem. As a result, sulfur is gradually being released or leached from the watershed into the surface waters as sulfate. Scientists now expect that the release of sulfate that previously accumulated in watersheds will delay the recovery of surface waters in the Northeast that is anticipated in response to the recent SO_2 emission controls (Driscoll et al., 2001).

A major study of the ecological response to acidification is taking place in the Bear Brook Watershed in Maine. Established in 1986 as part of the EPA's Watershed Manipulation Project, the project has found that experimental additions of sulfur and nitrogen to the watershed increased the concentrations of both sulfate and nitrate in the West Bear Brook stream. Stream water concentrations of several other ions, including base cations, aluminum, and ANC, changed substantially as well (Norton et al., 1999). During the first year of treatment, 94 percent of the nitrogen added experimentally to the Bear Brook watershed was retained, while the remainder leached into streams as nitrate. Nitrogen retention decreased to about 82 percent in subsequent years (Kahl et al., 1993; 1999). Although the forest ecosystem continued to accumulate nitrogen, nitrate leaching into the stream continued at elevated levels throughout the length of the experiment. This nitrate contributed to both episodic and chronic acidification of the stream. This and other similar studies have allowed scientists to quantify acidification and recovery relationships in eastern watersheds in much more detail than was possible in 1990.

The Appalachian Mountain region receives some of the highest rates of acid deposition in the United States (Herlihy et al., 1993). The acid-base status of stream waters in forested upland watersheds in the Appalachian Mountains was extensively investigated in the early 1990s (e.g., Church et al. [1992]; Herlihy et al. [1993]; Webb et al. [1994]; van Sickle and Church [1995]). A more recent assessment of the southern Appalachian region from West Virginia to Alabama identified watersheds that are sensitive to acid deposition using geologic bedrock and the associated buffering capacity of soils to neutralize acid. The assessment found that approximately 59 percent of all trout stream length in the region is in areas that are highly vulnerable to acidification and that 27 percent is in areas that are moderately vulnerable (SAMAB, 1996). Another study estimated that 18 percent of potential brook trout streams in the mid-Appalachian Mountains are too acidic for brook trout survival (Herlihy et al., 1996). Perhaps the most important study of acid-base chemistry of streams in the Appalachian region in recent years has been the Virginia Trout Stream Sensitivity Study (Webb et al., 1994). Trend analyses of these streams indicate that few long-term sampling sites are recovering from acidification, most are continuing to acidify, and the continuing acidification is at levels that are biologically significant for brook trout populations (Webb et al., 2000).

5.3.1.1 Water/Watershed Modeling

Researchers have used models to help them understand and predict atmospheric, environmental, and human health responses to acid deposition for well over 20 years. Since 1990, watershed modeling capabilities have also improved as researchers are continuing to refine and expand models that project acidification of waterbodies. Unlike the response of air quality and deposition to changes in emissions, lakes and streams take years to decades to fully reflect reductions in acid deposition. In some cases, soil chemistry has been significantly altered, and ions must either build up or be leached out before the chemistry can return to its pre-acidification status. Therefore, lake and stream conditions are presented for 2030. These results may still not reflect the full scope of ecosystem response to CAIR emissions reductions.

5.3.1.2 Description of the MAGIC Model and Methods

A number of mathematical models of soil and surface water acidification in response to atmospheric deposition were developed in the early 1980s (e.g., Christopherson and Wright [1981]; Christopherson et al. [1982]; Schnoor et al. [1984]; Booty and Kramer [1984]; Goldstein et al. [1984]; Cosby et al. [1985a,b,c]). These models were based on process-level information about the acidification process and were built for a variety of purposes ranging from estimating transient water quality responses for individual storm events to estimating chronic acidification of soils and base flow surface water. One of these models (MAGIC—the Model of Acidification of Groundwater In Catchments; Cosby et al. [1985a,b,c]) has been in use now for more than 15 years. MAGIC has been applied extensively in North America and Europe to both individual sites and regional networks of sites and has also been used in Asia, Africa, and South America. The utility of MAGIC for simulating a variety of water and soil acidification responses at the laboratory, plot, hillslope, and catchment scales has been tested using long-term monitoring data and experimental manipulation data. MAGIC has been widely used in policy and assessment activities in the United States and in several countries in Europe.

5.3.1.3 Model Structure

MAGIC is a lumped-parameter model of intermediate complexity, developed to predict the long-term effects of acidic deposition on surface water chemistry. The model simulates soil solution chemistry and surface water chemistry to predict the monthly and annual average concentrations of the major ions in these waters. MAGIC consists of the following: (1) a section in which the concentrations of major ions are assumed to be governed by simultaneous reactions involving sulfate adsorption, cation exchange, dissolution-precipitation-speciation of aluminum, and dissolution-speciation of inorganic carbon; and (2) a mass balance section in which the flux of major ions to and from the soil is assumed to be controlled by atmospheric inputs, chemical weathering, net uptake, and loss in biomass and losses to runoff. At the heart of MAGIC is the size of the pool of exchangeable base cations in the soil. As the fluxes to and from this pool change over time owing to changes in atmospheric deposition, the chemical equilibria between soil and soil solution shift to give changes in surface water chemistry. The degree and rate of change of surface water acidity thus depend both on flux factors and the inherent characteristics of the affected soils.

Cation exchange is modeled using equilibrium (Gaines-Thomas) equations with selectivity coefficients for each base cation and aluminum. Sulfate adsorption is represented

by a Langmuir isotherm. Aluminum dissolution and precipitation are assumed to be controlled by equilibrium with a solid phase of aluminum trihydroxide. Aluminum speciation is calculated by considering hydrolysis reactions as well as complexation with sulfate, fluoride, and dissolved organic compounds. Effects of carbon dioxide on pH and on the speciation of inorganic carbon are computed from equilibrium equations. Organic acids are represented in the model as tri-protic analogues. Weathering rates are assumed to be constant. Two alternative mechanisms are offered for simulating nitrate and ammonium in soils: either (1) first order equations representing net uptake and retention or (2) a set of equations and compartments describing process-based nitrogen dynamics in soils controlled by soil nitrogen pools. Input-output mass balance equations are provided for base cations and strong acid anions, and charge balance is required for all ions in each compartment. Given a description of the historical, current, and expected future deposition at a site, the model equations are solved numerically to give long-term reconstructions of surface water chemistry (for complete details of the model see Cosby et al. [1985 a,b,c]; [2001]).

MAGIC has been used to reconstruct the history of acidification, to examine current patterns of recovery, and to simulate the future trends in stream water acidity in both individual catchment and regional applications at a large number of sites across North America and Europe (e.g., Beier et al. [1995]; Cosby et al. [1985b,1990, 1995, 1996]; Cosby and Wright, 1998; Ferrier et al. [2001]; Hornberger et al. [1989]; Jenkins et al. [1990]; Moldan et al. [1998]; Norton et al. [1992]; Whitehead et al. [1988, 1997]; Wright et al. [1990, 1994, 1998]).

5.3.1.4 Model Implementation

Atmospheric deposition and net uptake-release fluxes for the base cations and strong acid anions are required as inputs to the model. These inputs are generally assumed to be uniform over the catchment. Atmospheric fluxes are calculated from concentrations of the ions in precipitation and the rainfall volume into the catchment. The atmospheric fluxes of the ions must be corrected for dry deposition of gas, particulates, and aerosols and for inputs in cloud/fog water. The volume discharge for the catchment must also be provided to the model. In general, the model is implemented using average hydrologic conditions and meteorological conditions in annual or seasonal simulations (i.e., mean annual or mean monthly deposition); precipitation and lake discharge are used to drive the model. Values for soil and surface water temperature, partial pressure of carbon dioxide, and organic acid concentrations must also be provided at the appropriate temporal resolution.

In this application, deposition input to MAGIC was estimated from the policy scenario emissions data based on previous known relationships between emissions of acid deposition precursors and acid deposition. As implemented in this project, the model is a two-compartment representation of a catchment. Atmospheric deposition enters the soil compartment, and the equilibrium equations are used to calculate soil water chemistry. The water is then routed to the stream compartment, and the appropriate equilibrium equations are reapplied to calculate runoff chemistry.

Once initial conditions (initial values of variables in the equilibrium equations) have been established, the equilibrium equations are solved for soil water and surface water concentrations of the remaining variables. These concentrations are used to calculate the lake discharge output fluxes of the model for the first time step. The mass balance equations are (numerically) integrated over the time step, providing new values for the total amounts of base cations and strong acid anions in the system. These in turn are used to calculate new values of the remaining variables, new lake discharge fluxes, and so forth. The output from MAGIC is thus a time trace for all major chemical constituents for the period of time chosen for the integration.

5.3.1.5 Calibration Procedure

The aggregated nature of the model requires that it be calibrated to observed data from a system before it can be used to examine potential system response. Calibration is achieved by setting the values of certain parameters within the model that can be directly measured or observed in the system of interest (called "fixed" parameters). The model is then run (using observed atmospheric and hydrologic inputs) and the simulated values of surface water and soil chemical variables (called "criterion" variables) are compared to observed values of these variables. If the observed and simulated values differ, the values of another set of parameters in the model (called "optimized" parameters) are adjusted to improve the fit. After a number of iterations, the simulated-minus-observed values of the criterion variables usually converge to zero (within some specified tolerance). The model is then considered calibrated. If new assumptions (or values) for any of the fixed variables or inputs to the model are subsequently adopted, the model must be recalibrated by readjusting the optimized parameters until the simulated-minus-observed values of the criterion variables again fall within the specified tolerance.

Calibrations are based on volume-weighted mean annual or seasonal fluxes for a given period of observation. The length of the period of observation used for calibration is not arbitrary. Model output will be more reliable if the annual flux estimates used in

calibration are based on a number of years rather than just 1 year. There is a lot of year-toyear variability in atmospheric deposition and catchment runoff. Averaging over a number of years reduces the likelihood that an "outlier" year (very dry, etc.) is the primary data on which model forecasts are based. On the other hand, averaging over too long a period may remove important trends in the data that the model needs to simulate.

The calibration procedure requires that stream water quality, soil chemical and physical characteristics, and atmospheric deposition data be available for each catchment. The water quality data needed for calibration are the concentrations of the individual base cations (Ca, Mg, Na, and K) and acid anions (Cl, SO₄, and NO₃) and the pH. The soil data used in the model include soil depth and bulk density, soil pH, soil cation-exchange capacity, and exchangeable bases on the soil (Ca, Mg, Na, and K). The atmospheric deposition inputs to the model must be estimates of total deposition, not just wet deposition. In some instances, direct measurements of either atmospheric deposition or soil properties may not be available for a given site with stream water data. In these cases, the required data can often be estimated by assigning soil properties based on some landscape classification of the catchment and assigning deposition using model extrapolations from some national or regional atmospheric deposition monitoring network.

Soil Physical and Chemical Properties. Soils data for model calibration are usually derived as averaged values of soil parameters within a catchment. If soils data for a given location are vertically stratified, the soils data for the individual soil horizons at that sampling site can be aggregated based on horizon, depth, and bulk density to obtain single vertically aggregated values for the site, or the stratified data can be used directly in the model.

Total Atmospheric Deposition. Total atmospheric deposition consists of three components: wet deposition, the flux of ions occurring in precipitation; dry deposition, resulting from gaseous and particulate fluxes; and cloud/fog deposition (which can be particularly important in mountainous inland areas or moderate highlands in areas adjacent to oceans or seas). Estimates of precipitation volume and ionic concentrations in precipitation can be used to calculate wet deposition for a site. Observations of dry deposition or cloud/fog deposition are very infrequent. The approach usually used to quantify these components relies on some estimate of the ratio of estimated total deposition to the observed wet deposition for important ions (e.g., sulphate, nitrate, and ammonium ions). These ratios (called dry deposition factors) are then used to calculate total deposition from the observed wet deposition data. *Historical Loading*. Calibration of the model (and estimation of the historical changes at the sites) requires a temporal sequence of historical anthropogenic deposition. Our current understanding of ecosystem responses to acidic deposition suggests that future ecosystem responses can be strongly conditioned by historical acidic loadings. Thus, as part of the model calibration process, the model should be constrained by some measure of historical deposition to the site. However, such long-term, continuous historical deposition data do not exist. The usual approach is to use historical emissions data as a surrogate for deposition. The emissions for each year in the historical period can be normalized to emissions in a reference year (a year for which observed deposition data are available). Using this scaled sequence of emissions, historical deposition can be estimated by multiplying the total deposition estimated for each site in reference year by the emissions scale factor for any year in the past to obtain deposition for that year.

5.3.1.6 MAGIC Modeling Results

Watershed modeling was undertaken for the CAIR proposal. It was determined that the watershed modeling conducted for the proposal is essentially unchanged for the final rule. This modeling projects that 1 percent of northeastern lakes would be chronically acidic in 2030 as a result of CAIR. In contrast, the same model used to analyze existing control programs projects 6 percent of northeastern lakes would be chronically acidic in 2030. The modeling projects that, under CAIR, 28 percent of northeastern lakes would be episodically acidic in 2030, compared to 25 percent in 2030 under existing control programs. For Adirondack lakes, a subset of northeastern lakes, the signals of surface water chemical recovery are much stronger. Under CAIR, no Adirondack lakes would be chronically acidic, and 64 percent would be episodically acidic in 2030, as opposed to 12 percent chronically acidic, acidic and 52 percent episodically acidic in 2030 under current control programs.

Because of the age and types of soils in many high-elevation areas of the southeast, streams in that region are more frequently characterized by a delayed response to changes in deposition. For the ecosystems modeled in this region, 17 percent of streams are currently chronically acidic, and this level stays the same under CAIR in 2030; the proportion of episodically acidic streams increases from 19 percent under current conditions to 23 percent under CAIR, which reflects a decrease in the proportion of nonacidic streams from 64 percent under current conditions to 60 percent under CAIR in 2030. It is important to note that, under the base case, the proportion of nonacidic streams decreases even further, dropping from 64 percent under current conditions to 58 percent in 2030. Thus, in the southeast, CAIR would slow the deterioration of stream health (episodically acidic) expected

under the base case and would prevent additional streams from becoming chronically acidic. Results of the MAGIC modeling are summarized in Table 5-1.

	Current	Base Case (2030)	Transport Rule (2030)
Northeastern Lakes			
chronically acidic	10%	6%	1%
episodically acidic	21%	25%	28%
nonacidic	69%	69%	71%
Adirondack Lakes			
chronically acidic	21%	12%	0%
episodically acidic	43%	52%	64%
nonacidic	36%	36%	36%
Southeastern Streams			
chronically acidic	17%	17%	17%
episodically acidic	19%	25%	23%
nonacidic	64%	58%	60%

Table 5-1. Acidification Changes in Water Bodies as a Result of CAIR

5.3.1.7 Study of Benefits of Natural Resource Improvements in the Adirondacks

The EPA conducted MAGIC modeling to quantify acidification improvements likely to occur as a result of CAIR in lakes in the northeastern U.S., the Adirondack Lakes, and streams in the southeastern U.S. However, we were unable to estimate the monetary benefits associated with the improvements predicted by the MAGIC modeling. A study conducted by RFF estimates the monetary benefits associated with natural resource improvements due to acidification reductions in the Adirondacks (Banzhaf, 2004). Since CAIR results in acidification improvements in the Adirondack Lakes, the RFF study may be relevant to this rulemaking. The RFF study estimates the total benefits (i.e., the sum of use and nonuse values) of natural resource improvements for the Adirondacks resulting from a program that **US EPA ARCHIVE DOCUMENT**

would reduce acidification in 40 percent of the lakes in the Adirondacks of concern for acidification. RFF estimates that there are significant benefits for the hypothesized improvements in lake health for New York State households. The study is undergoing peer review with an intent to publish in the near future. Based on the peer review, the Agency will determine whether and how it should be used to quantify the benefits associated with reductions in acidification. As the MAGIC modeling indicates CAIR is also expected to result in significant acid deposition reductions for the Adirondacks, leading to reduced acidification and improved lake health. The benefits of these improvements in lake health relative to the RFF estimates will depend upon the exact magnitude of the acid deposition reductions, the number of lakes with improvements, and the magnitude of acidification improvements. The RFF study suggests that the benefits of acid deposition reductions for CAIR could be substantial in terms of the total monetized value for ecological endpoints.

5.3.2 Forest Ecosystems

Reductions in sulfur and nitrogen deposition under CAIR are expected to reduce the effects of acid deposition on forests. Our current understanding of the effects of acid deposition on forest ecosystems has come to focus increasingly on the effects of biogeochemical processes that affect plant uptake, retention, and cycling of nutrients within forested ecosystems. Research results from the 1990s indicate that documented decreases in base cations (calcium, magnesium, potassium, and others) from soils in the northeastern and southeastern United States are at least partially attributable to acid deposition (Lawrence et al., 1997; Huntington et al., 2000). Base cation depletion is a cause for concern because of the role these ions play in acid neutralization and, in the case of calcium, magnesium, and potassium, their importance as essential nutrients for tree growth. It has been known for some time that depletion of base cations from the soil interferes with the uptake of calcium by roots in forest soils (Shortle and Smith, 1988). Recent research indicates it also leads to aluminum mobilization (Lawrence et al., 1995), which can have harmful effects on fish (Dept. of Interior, 2003).

The plant physiological processes affected by reduced calcium availability include cell wall structure and growth, carbohydrate metabolism, stomatal regulation, resistance to plant pathogens, and tolerance of low temperatures (DeHayes et al., 1999). Soil structure, macro and micro fauna, decomposition rates, and nitrogen metabolism are also important processes that are significantly influenced by calcium levels in soils. The importance of calcium as an indicator of forest ecosystem function is due to its diverse physiological roles, coupled with the fact that calcium mobility in plants is very limited and can be further reduced by tree age, competition, and reduced soil water supply (McLaughlin and Wimmer, 1999).

A clear link has now been established in red spruce stands between acid deposition, calcium supply, and sensitivity to abiotic stress. Red spruce uptake and retention of calcium is affected by acid deposition in two main ways: leaching of important stores of calcium from needles (DeHayes et al., 1999) and decreased root uptake of calcium due to calcium depletion from the soil and aluminum mobilization (Smith and Shortle, 2001; Shortle et al., 1997; Lawrence et al., 1997). Acid deposition leaches calcium from mesophyll cells of 1-year-old red spruce needles (Schaberg et al., 2000), which in turn reduces freezing tolerance (DeHayes et al., 1999). These changes increase the sensitivity of red spruce to winter injuries under normal winter conditions in the Northeast, result in the loss of needles, and impair the overall health of forest ecosystems (DeHayes et al., 1999). Red spruce must also expend more metabolic energy to acquire calcium from soils in areas with low calcium/aluminum ratios, resulting in slower tree growth (Smith and Shortle, 2001).

Losses of calcium from forest soils and forested watersheds have now been documented as a sensitive early indicator of the soil response to acid deposition for a wide range of forest soils in the United States (Lawrence et al., 1999; Huntington et al., 2000). There is a strong relationship between acid deposition and leaching of base cations from hardwood forest (e.g., maple, oak) soils, as indicated by long-term data on watershed mass balances (Likens et al., 1996; Mitchell et al., 1996), plot- and watershed-scale acidification experiments in the Adirondacks (Mitchell et al., 1994) and in Maine (Norton et al., 1994; Rustad et al., 1996), and studies of soil solution chemistry along an acid deposition gradient from Minnesota to Ohio (MacDonald et al., 1992).

Although sulfate is the primary cause of base cation leaching, nitrate is a significant contributor in watersheds that are nearly nitrogen saturated (Adams et al., 1997). Recent studies of the decline of sugar maples in the Northeast demonstrate a link between low base cation availability, high levels of aluminum and manganese in the soil, and increased levels of tree mortality due to native defoliating insects (Horsley et al., 2000). The chemical composition of leaves and needles may also be altered by acid deposition, resulting in changes in organic matter turnover and nutrient cycling.

5.3.3 Coastal Ecosystems

Since 1990, a large amount of research has been conducted on the impact of nitrogen deposition to coastal waters. It is now known that nitrogen deposition is a significant source of nitrogen to many estuaries (Valigura et al., 2001; Howarth, 1998). The amount of nitrogen entering estuaries due to atmospheric deposition varies widely, depending on the size and location of the estuarine watershed and other sources of nitrogen in the watershed. For a handful of estuaries, atmospheric deposition of nitrogen contributes well over 40 percent of the total nitrogen load; however, in most estuaries for which estimates exist, the contribution from atmospheric deposition ranges from 15 to 30 percent. The area with the highest deposition rates (30 percent deposition rates) stretches from Massachusetts to the Chesapeake Bay and along the central Gulf of Mexico coast.

Nitrogen is often the limiting nutrient in coastal ecosystems. Increasing the levels of nitrogen in coastal waters can cause significant changes to those ecosystems. Approximately 60 percent of estuaries in the United States (65 percent of the estuarine surface area) suffer from overenrichment of nitrogen, a condition known as eutrophication (Bricker et al., 1999). Symptoms of eutrophication include changes in the dominant species of plankton (the primary food source for many kinds of marine life) that can cause algal blooms, low levels of oxygen in the water column, fish and shellfish kills, and cascading population changes up the food chain. Many of the most highly eutrophic estuaries are along the Gulf and mid-Atlantic coasts, overlapping many of the areas with the highest nitrogen deposition, but there are eutrophic estuaries in every region of the coterminous U.S. coastline.

Figure 5-4 shows the percent reductions in nitrogen deposition to coastal hydrologic regions expected to result from CAIR within the CAIR region. As Figure 5-4 depicts, changes in all forms of nitrogen deposition in the hydrologic regions within the CAIR region are expected to range from small areas of slight increases that do not exceed 1.5 percent to reductions up to 19 percent and average reductions of 4.6 percent for areas east of the 100th parallel in 2015.

The Chesapeake Bay Program estimated the reduced mass of delivered nitrogen loads likely to result from CAIR, based upon the CAIR proposal deposition estimates published in January 2004 (Sweeney, 2004). Atmospheric deposition of nitrogen accounts for a significant portion of the nitrogen loads to the Chesapeake with 28 percent of the nitrogen loads from the watershed coming from air deposition. Based upon the CAIR proposal nitrogen deposition rates published in the January 2004 proposal, the Chesapeake Bay

2015 Percent Reductions of Nitrogen To Coastal Hydrologic Regions Under CAIR (compared

with a projected 2015 base case)

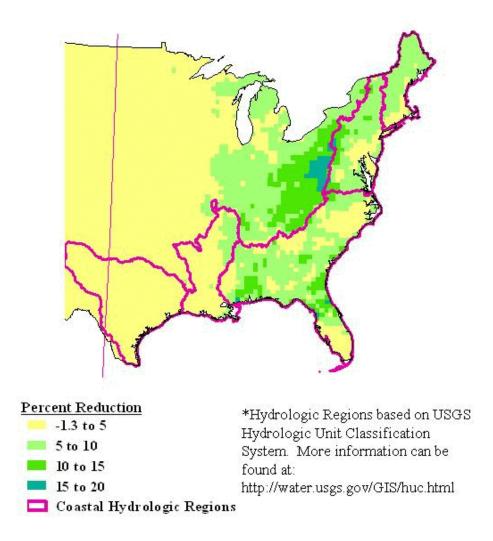


Figure 5-4. CAIR Nitrogen Deposition Reductions in Hydrologic Regions

Program finds that CAIR will likely reduce the nitrogen loads to the Bay by 10 million pounds per year by 2010. Nitrogen deposition reductions for the final CAIR are anticipated to be comparable to the proposed rule in this region. These substantial nitrogen load reductions more than fulfill the EPA's commitment to reduce atmospheric deposition delivered to the Chesapeake Bay by 8 million pounds annually. The monetized value of such reductions for the Bay are likely to be substantial, but we are unable to estimate the monetary value of these reductions at this time.

5.3.4 Potential Other Impacts

This rule is expected to result in many categories of benefits that we are currently unable to quantify or monetize. It is possible that reductions in nitrogen deposition resulting from this rule may lessen the benefits of passive fertilization for forests and terrestrial ecosystems where nutrients are a limiting factor and for some croplands.

The effects of ozone and particulate matter on radiative transfer in the atmosphere can also lead to effects of uncertain magnitude and direction on the penetration of ultraviolet light and climate. Ground level ozone makes up a small percentage of total atmospheric ozone (including the stratospheric layer) that attenuates penetration of UVb radiation to the ground. EPA's past evaluation of the information indicates that potential disbenefits would be small, variable, and with too many uncertainties to attempt quantification of relatively small changes in average ozone levels over the course of a year (EPA, 2005a). EPA's most recent provisional assessment of the currently available information indicates that potential but unquantifiable benefits may also arise from reducing ozone-related attenuation of UVb radiation (EPA, 2005b). Sulfate and nitrate particles also scatter UVb, which can decrease exposure of horizontal surfaces to UVb, but increase exposure of vertical surfaces. In this case as well, both the magnitude and direction of the effect of reductions in sulfate and nitrate particles are too uncertain to quantify (EPA, 2004). Ozone is a greenhouse gas, and sulfates and nitrates can reduce the amount of solar radiation reaching the earth, but EPA believes that we are unable to quantify any net climate-related disbenefit or benefit associated with the combined ozone and PM reductions in this rule.

5.4 References

Adams, M.B., T.R. Angradi, and J.N. Kochenderfer. 1997. "Stream Water and Soil Solution Responses to 5 Years of Nitrogen and Sulfur Additions at the Fernow Experimental Forest, West Virginia." *Forest Ecology and Management* 95:79-91.

- Banzhaf, Spencer, Dallas Burtraw, David Evans, and Alan Krupnick. September 2004. "Valuation of Natural Resource Improvements in the Adirondacks." Resources for the Future (RFF).
- Beier, C., H. Hultberg, F. Moldan, and Wright. 1995. "MAGIC Applied to Roof Experiments (Risdalsheia, N.; Gårdsjön, S.; Klosterhede, D.K.) to Evaluate the Rate of Reversibility of Acidification Following Experimentally Reduced Acid Deposition." *Water Air Soil Pollution* 85:1745-1751.
- Booty, W.G. and J.R. Kramer. 1984. "Sensitivity Analysis of a Watershed Acidification Model." *Philos. Trans. R. Soc. London, Ser. B* 305:441-449.
- Bricker, S.B., C.G. Clement, D.E. Pirhalla, S.P. Orlando, and D.R.G. Farrow. 1999.
 National Estuarine Eutrophication Assessment: Effects of Nutrient Enrichment in the Nation's Estuaries. National Oceanic and Atmospheric Administration, National Ocean Service, Special Projects Office and the National Centers for Coastal Ocean Science. Silver Spring, Maryland.
- Christopherson, N., and R.F. Wright. 1981. "Sulfate Budget and a Model for Sulfate Concentrations in Streamwater at Birkenes, a Small Forested Catchment in Southernmost Norway." *Water Resources Research* 17:377–389.
- Christopherson, N., H.M. Seip, and R.F. Wright. 1982. "A Model for Streamwater Chemistry at Birkenes, Norway." *Water Resources Research* 18:977–996.
- Church, M.R., P.W. Shaffer, K.W. Thornton, D.L. Cassell, C.I. Liff, M.G. Johnson, D.A. Lammers, J.J. Lee, G.R. Holdren, J.S. Kern, L.H. Liegel, S.M. Pierson, D.L. Stevens, B.P. Rochelle, and R.S. Turner. 1992. *Direct/Delayed Response Project: Future Effects of Long-Term Sulfur Deposition on Stream Chemistry in the Mid-Appalachian Region of the Eastern United States*. U.S. Environmental Agency, EPA/600/R-92/186, Washington, DC. 384 pp.
- Cosby, B.J., G.M. Hornberger, R.F. Wright, E.B. Rastetter, and J.N. Galloway. 1995.
 "Estimating Catchment Water Quality Response to Acid Deposition Using Mathematical Models of Soil Ion Exchange Processes." *Geoderma* 38:77-95.
- Cosby, B.J. and R.F. Wright. 1998. "Modelling Regional Response of Lakewater Chemistry to Changes in Acid Deposition: The MAGIC Model Applied to Lake Surveys in Southernmost Norway 1974-1986-1995." *Hydrology and Earth Systems Sciences* 2:563-576.

- Cosby, B.J., R.F. Wright, G.M. Hornberger, and J.N. Galloway. 1985a. Modelling the Effects of Acid Deposition: Assessment of a Lumped Parameter Model of Soil Water and Streamwater Chemistry." *Water Resources Research* 21:51-63.
- Cosby, B.J., R.F. Wright, G.M. Hornberger, and J.N. Galloway. 1985b. "Modelling the Effects of Acid Deposition: Estimation of Long-Term Water Quality Responses in a Small Forested Catchment." *Water Resources Research* 21:1591-1601.
- Cosby, B.J., G.M. Hornberger, J.N. Galloway, and R.F. Wright. 1985c. "Time Scales of Catchment Acidification: A Quantitative Model for Estimating Freshwater Acidification." *Environmental Science Technology* 19:1144-1149.
- Cosby, B.J., A. Jenkins, J.D. Miller, R.C. Ferrier, and T.A.B. Walker. 1990. "Modelling Stream Acidification in Afforested Catchments: Long-Term Reconstructions at Two Sites in Central Scotland." *Journal of Hydrology* 120:143-162.
- Cosby, B.J., R.F. Wright, and E. Gjessing. 1995. "An Acidification Model (MAGIC) with Organic Acids Evaluated Using Whole-Catchment Manipulations in Norway." *Journal of Hydrology* 170:101-122.
- Cosby, B.J., R.C. Ferrier, A. Jenkins, and R.F. Wright. 2001. "Modeling the Effects of Acid Deposition: Refinements, Adjustments and Inclusion of Nitrogen Dynamics in the MAGIC Model." *Hydrology and Earth Systems Sciences* 5:499-517.
- DeHayes, D.H., P.G. Schaberg, G.J. Hawley, and G.R. Strimbeck. 1999. "Acid Rain Impacts Calcium Nutrition and Forest Health: Alteration of Membrane-Associated Calcium Leads to Membrane Destabilization and Foliar Injury in Red Spruce." *Bioscience* 49:789-800.
- Driscoll, C.T., G. Lawrence, A. Bulger, T. Butler, C. Cronan, C. Eagar, K.F. Lambert, G.E. Likens, J. Stoddard, and K. Weathers. 2001. "Acid Deposition in the Northeastern U.S.: Sources and Inputs, Ecosystem Effects, and Management Strategies." *Bioscience* 51:180-198.
- Ferrier, R.C., R.C. Helliwell, B.J. Cosby, A. Jenkins, and R.F. Wright. 2001. "Recovery from Acidification of Lochs in Galloway, South-west Scotland, UK: 1979-1998." *Hydrology and Earth System Sciences* 5:421-431.
- Goldstein, R.A., S.A. Gherini, C.W. Chen, L. Mak, and R.J.M. Hudson. 1984. "Integrated Acidification Study (ILWAS): A Mechanistic Ecosystem Analysis." *Phil. Trans. R. Soc. London, Ser. B*, 305:409-425.

- Herlihy, A.T., P.R. Kaufmann, M.R. Church, P.J. Wigington, Jr., J.R. Webb, and M.J. Sale.
 1993. "The Effects of Acid Deposition on Streams in the Appalachian Mountain and Piedmont Region of the Mid-Atlantic United States." *Water Resources Research* 29:2687-2703.
- Herlihy, A.T., P.R. Kaufmann, J.L. Stoddard, K.N. Eshleman, and A.J. Bulger. 1996. Effects of Acidic Deposition on Aquatic Resources in the Southern Appalachians with a Special Focus on Class I Wilderness Areas. The Southern Appalachian Mountain Initiative (SAMI).
- Hornberger, G.M., B.J. Cosby, and R.F. Wright. 1989. "Historical Reconstructions and Future Forecasts of Regional Surface Water Acidification in Southernmost Norway." *Water Resources Research* 25:2009-2018.
- Horsley, S.B., R.P. Long, S.W. Bailey, R.A. Hallett, and T.J. Hall. 2000. "Factors Associated with the Decline Disease of Sugar Maple on the Allegheny Plateau." *Canadian Journal of Forest Research* 30:1365-1378.
- Howarth, Robert. 1998. "An Assessment of Human Influences on Fluxes of Nitrogen from the Terrestrial Landscape to the Estuaries and Continental Shelves of the North Atlantic Ocean." Nutrient Cycling in Agroecosystems 52(2/3):213-223.
- Huntington, T.G., R.P. Hooper, C.E. Johnson, B.T. Aulenbach, R. Cappellato, and A.E. Blum. 2000. "Calcium Depletion in a Southeastern United States Forest Ecosystem." *Soil Science Society of America Journal* 64:1845-1858.
- Jenkins, A., P.G. Whitehead, B.J. Cosby, and H.J.B. Birks. 1990. "Modelling Long-Term Acidification: A Comparison with Diatom Reconstructions and the Implications for Reversibility." *Phil. Trans. R. Soc. London B* 327:435-440.
- Kahl, J., S. Norton, I. Fernandez, L. Rustad, and M. Handley. 1999. "Nitrogen and Sulfur Input-Output Budgets in the Experimental and Reference Watersheds, Bear Brook Watershed, Maine (BBWM)." *Environmental Monitoring and Assessment* 55:113-131.
- Kahl, J.S., S.A. Norton, I.J. Fernandez, K.J. Nadelhoffer, C.T. Driscoll, and J.D. Aber. 1993.
 "Experimental Inducement of Nitrogen Saturation at the Watershed Scale." *Environmental Science and Technology* 27:565-568.
- Lawrence, G.B., M.B. David, and W.C. Shortle. 1995. "A New Mechanism for Calcium Loss in Forest-Floor Soils." *Nature* 378:162-165.

- Lawrence, G.W., M.B. David, S.W. Bailey, and W.C. Shortle. 1997. "Assessment of Calcium Status in Soils of Red Spruce Forests in the Northeastern United States." *Biogeochemistry* 38:19-39.
- Lawrence, G.B., M.B. David, G.M. Lovett, P.S. Murdoch, D.A. Burns, J.L. Stoddard, B.P. Baldigo, J.H. Porter, and A.W. Thompson. 1999. "Soil Calcium Status and the Response of Stream Chemistry to Changing Acidic Deposition Rates in the Catskill Mountains of New York." *Ecological Applications* 9:1059-1072.
- Likens, G.E., C.T. Driscoll, and D.C. Buso. 1996. "Long-Term Effects of Acid Rain: Responses and Recovery of a Forest Ecosystem." *Science* 272:244-246.
- MacDonald, N.W., A.J. Burton, H.O. Liechty, J.A. Whitter, K.S. Pregitzer, G.D. Mroz, and D.D. Richter. 1992. "Ion Leaching in Forest Ecosystems along a Great Lakes Air Pollution Gradient." *Journal of Environmental Quality* 21:614-623.
- McLaughlin S.B. and R. Wimmer. 1999. "Tansley Review No. 104, Calcium Physiology and Terrestrial Ecosystem Processes." *New Phytologist* 142:373-417.
- Mitchell, M.J., M.B. David, I.J. Fernandez, R.D. Fuller, K. Nadelhoffer, L.E. Rustad, and A.C. Stam. 1994. "Response of Buried Mineral Soil Bags to Experimental Acidification of Forest Ecosystem." *Soil Science Society of America Journal* 58:556-563.
- Mitchell, M.J., C.T. Driscoll, J.S. Kahl, G.E. Likens, P.S. Murdoch, and L.H. Pardo. 1996. "Climate Control on Nitrate Loss from Forested Watersheds in the Northeast United States." *Environmental Science and Technology* 30:2609-2612.
- Moldan, F., R.F. Wright, R.C. Ferrier, B.I., Andersson, and H. Hultberg. 1998. "Simulating the Gårdsjön Covered Catchment Experiment with the MAGIC Model." In *Experimental Reversal of Acid Rain Effects. The Gårdsjön Roof Project*, Hultberg, H. and Skeffington, R.A. (eds.), p. 351-362, Chichester, UK: Wiley and Sons, 466 pp.
- Murdoch, P.S., D.S. Burns, and G.B. Lawrence. 1998. "Relation of Climate Change to the Acidification of Surface Waters by Nitrogen Deposition." *Environmental Science and Technology* 32:1642-1647.
- National Acid Precipitation Assessment Program (NAPAP). 1991. 1990 Integrated Assessment Report. Washington, DC: National Acid Precipitation Assessment Program Office of the Director.

- Norton, S.A. R.F. Wright, J.S. Kahl, and J.P. Scofield. 1992. "The MAGIC Simulation of Surface Water Acidification at, and Preliminary Results from, the Bear Brook Watershed Manipulation, Maine." *Environmental Pollution* 77:279-286.
- Norton, S.A., J.S. Kahl, I.J. Fernandez, L.E. Rustad, J.P. Schofield, and T.A. Haines. 1994.
 "Response of the West Bear Brook Watershed, Maine, USA, to the addition of (NH4)2SO4: 3-Year Results." *Forest and Ecology Management* 68:61-73.
- Norton, S.A., R.F. Wright, J.S. Kahl, and J.P. Scofield. 1998. "The MAGIC Simulation of Surface Water Acidification at, and First Year Results from, the Bear Brook Watershed Manipulation, Maine, USA." *Environmental Pollution* 77:279-286.
- Norton, S.A., J.S. Kahl, I.J. Fernandez, T.A. Haines, L.E. Rustad, S. Nodvin, J.P. Scofield, T. Strickland, H. Erickson, P. Wiggington, and J. Lee. 1999. "The Bear Brook Watershed, Maine, (BBWP) USA." *Environmental Monitoring and Assessment* 55:7-51.
- Rustad, L.E., I.J. Fernandez, M.B. David, M.J. Mitchell, K.J. Nadelhoffer, and R.D. Fuller. 1996. "Experimental Soil Acidification and Recovery at the Bear Brook Watershed in Maine." *Soil Science of America Journal* 60:1933-1943.
- Schaberg, P.G., D.H. DeHayes, G.J. Hawley, G.R. Strimbeck, J.R. Cumming, P.F. Murakami, and C.H. Borer. 2000. "Acid Mist, Soil Ca and Al Alter the Mineral Nutrition and Physiology of Red Spruce." *Tree Physiology* 20:73-85.
- Schnoor, J.L., W.D. Palmer, Jr., and G.E. Glass. 1984. "Modeling Impacts of Acid Precipitation for Northeastern Minnesota." In *Modeling of Total Acid Precipitation Impact* Schnoor, J.L. (ed.), pp. 155-173. Boston: Butterworth.
- Smith, K.T. and W.C. Shortle. 2001. "Conservation of Element Concentration in Xylem Sap of Red Spruce." *Trees* 15:148-153.
- Shortle, W.C. and K.T. Smith. 1988. "Aluminum-Induced Calcium Deficiency Syndrome in Declining Red Spruce Trees." *Science* 240:1017-1018.
- Shortle, W.C., K.T. Smith, R. Minocha, G.B. Lawrence, and M.B. David. 1997. "Acidic Deposition, Cation Mobilization, and Biochemical Indicators of Stress in Healthy Red Spruce." *Journal of Environmental Quality* 26:871-876.

Southern Appalachian Man and the Biosphere (SAMAB). 1996. *The Southern Appalachian Assessment: Summary Report*. Atlanta, GA: U.S. Department of Agriculture, Forest Service, Southern Region.

Sweeney, Jeff. "EPA's Chesapeake Bay Program Air Strategy." October 26, 2004.

- U.S. Department of the Interior, National Park Service. 2003. Assessment of Air Quality and Related Values in Shenandoah National Park. Technical Report NPS/NERCHAL/NRTR-03/090. Philadelphia, PA: National Park Service, Northeast Region. http://www.nps.gov/shen/air_quality.htm.
- U.S. Environmental Protection Agency (EPA). 2004. Air Quality Criteria for Particulate Matter (October 2004). http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm? deid=87903>.
- U.S. Environmental Protection Agency (EPA). 2005a. Air Quality Criteria for Ozone and Related Photochemical Oxidants (First External Review Draft). http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=114523>.
- U.S. Environmental Protection Agency (EPA). 2005b. Draft Air Quality Criteria for Ozone and Related Photochemical Oxidants E-Docket No. ORD-2004-0015 [Federal Register: January 31, 2005 (Volume 70, Number 19)] http://www.epa.gov/fedrgstr/EPA-AIR/2005/January/Day-31/a1720.htm>.
- Valigura, R.A., R.B. Alexander, M.S. Castro, T.P. Meyers, H.W. Paerl, P.E. Stacy, and R.E. Turner. 2001. Nitrogen Loading in Coastal Water Bodies: An Atmospheric Perspective. Washington, DC: American Geophysical Union.
- Van Sickle, J. and M.R. Church. 1995. Methods for Estimating the Relative Effects of Sulfur and Nitrogen Deposition on Surface Water Chemistry. EPA/600/R-95/172.
 Washington, DC: U.S. Environmental Protection Agency.
- Webb, J.R., F.A. Deviney, Jr., B.J. Cosby, A.J. Bulger, and J.N. Galloway. 2000. Change in Acid- Base Status in Streams in the Shenandoah National Park and the Mountains of Virginia. American Geophysical Union, Biochemical Studies of the Shenandoah National Park. http://www.nps.gov/shen/air_quality.htm.

- Webb, J.R., F.A. Deviney, J.N. Galloway, C.A. Rinehart, P.A Thompson, and S. Wilson. 1994. The Acid-Base Status of Native Brook Trout Streams in the Mountains of Virginia. A Regional Assessment Based on the Virginia Trout Stream Sensitivity Study. Charlottesville, VA: Univ. of Virginia. http://www.nps.gov/shen/ air_quality.htm.
- Whitehead, P.G., B. Reynolds, G.M. Hornberger, C. Neal, B.J. Cosby, and P. Paricos. 1988. "Modelling Long-Term Stream Acidification Trends in Upland Wales at Plynlimon." *Hydrological Processes* 2:357-368.
- Whitehead, P.G., J. Barlow, E.Y. Haworth, and J.K. Adamson. 1997. "Acidification in Three Lake District Tarns: Historical Long Term Trends and Modelled Future Behaviour under Changing Sulphate and Nitrate Deposition." *Hydrology and Earth System Sciences* 1:197-204.
- Wright, R.F., B.J. Cosby, M.B. Flaten, and J.O. Reuss. 1990. "Evaluation of an Acidification Model with Data from Manipulated Catchments in Norway." *Nature* 343:53-55.
- Wright, R.F., B.J. Cosby, R.C. Ferrier, A Jenkins, A.J. Bulger, and R. Harriman. 1994.
 "Changes in the Acidification of Lochs in Galloway, Southwestern Scotland, 1979-1988: The MAGIC Model Used to Evaluate the Role of Afforestation, Calculate Critical Loads, and Predict Fish Status." *Journal of Hydrology* 161:257-285.
- Wright, R.F., B.A. Emmett, and A. Jenkins. 1998. "Acid Deposition, Land-Use Change and Global Change: MAGIC7 Model Applied to Risdalsheia, Norway (RAIN and CLIMEX projects) and Aber, UK (NITREX project)." *Hydrology and Earth System Sciences* 2:385-397.

CHAPTER 6

ELECTRIC POWER SECTOR PROFILE

This chapter discusses important aspects of the power sector as they relate to CAIR, including the types of power-sector sources affected by CAIR, and provides background on the power sector and EGUs. In addition, this chapter provides some historical background on EPA regulation of and future projections for the power sector.

6.1 **Power-Sector Overview**

The functions of the power sector can be separated into three distinct operating activities: generation, transmission, and distribution.

6.1.1 Generation

Electricity generation is the first process in the delivery of electricity to consumers. The process of generating electricity, in most cases, involves creating heat to rotate turbines which, in turn, create electricity. The power sector consists of over 16,000 generating units, consisting of fossil-fuel fired units, nuclear units, and hydroelectric and renewable sources dispersed throughout the country (see Table 6-1).

Energy Source	Number of Generators	Generator Nameplate Capacity (MW)
Coal	1,566	338,199
Petroleum	3,076	43,206
Natural Gas	2,890	194,968
Dual Fired	2,974	180,174
Other Gases	104	2,210
Nuclear	104	104,933
Hydroelectric	4,157	96,343
Other Renewables	1,501	18,797
Other	41	756
Total	16.413	979,585

Table 6-1.	Existing	Electricity	Generating	Capacity	bv F	Energy	Source, 20	02
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Source: EIA

These electric-generating sources provide electricity for commercial, industrial, and residential uses, each of which consumes roughly one-third of the total electricity produced (see Table 6-2).

Table 6-2. Total U.S. Electric Power Industry Retail Sales in 2003 (Billion kWh)

	Ν	%
Residential	1,280	37%
Commercial	1,119	32%
Industrial	991	28%
Other	109	3%
All Sectors	3,500	100%

Source: EIA

In 2003, electric-generating sources produced 3,848 billion kWh to meet electricity demand. Roughly 70 percent of this electricity was produced through the combustion of fossil fuels, primarily coal and natural gas, with coal accounting for more than half of the total (see Table 6-3).

 Table 6-3. Electricity Net Generation in 2003 (Billion kWh)

	N	%
Coal	1,970	51%
Petroleum	118	3%
Natural Gas	629	16%
Other Gases	11	0.3%
Nuclear	764	20%
Hydroelectric	275	7%
Other	81	2%
Total	3,848	100%

Source: EIA

Note: Retail sales and net generation may not correspond exactly because net generation data may include net exported electricity and loss of electricity.

Coal-fired generating units typically supply "base-load" electricity, which means these units operate continuously throughout the day. Coal-fired generation, along with nuclear generation, meet the part of demand that is relatively constant. Gas-fired generation, however, typically supplies "peak" power, when there is increased demand for electricity (e.g., when businesses operate throughout the day or when people return home from work and run appliances and heating/air-conditioning, versus late at night or very early morning when demand for electricity is reduced).

### 6.1.2 Transmission

Transmission is the term used to describe the movement of electricity, through use of high voltage lines, from electric generators to substations where power is stepped down for local distribution. Transmission systems have been traditionally characterized as a collection of independently operated networks or grids interconnected by bulk transmission interfaces.

Within a well-defined service territory, the regulated utility has historically had responsibility for all aspects of developing, maintaining, and operating transmission of electricity. These responsibilities typically included system planning and expanding, maintaining power quality and stability, and responding to failures.

### 6.1.3 Distribution

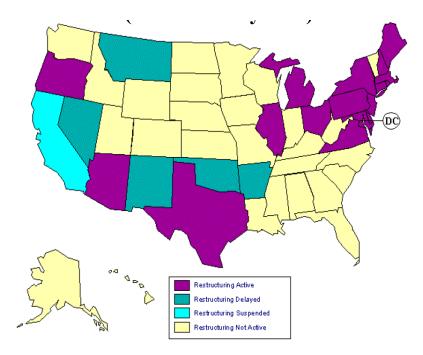
Distribution of electricity involves networks of smaller wires and substations that take the higher voltage from the transmission system and step it down to lower levels to match the needs of customers. The transmission and distribution system is the classic example of a natural monopoly because it is not practical to have more than one set of lines running from the electricity-generating sources to neighborhoods or from the curb to the house.

Transmission and distribution have been considered differently than generation in current efforts to restructure the industry. Transmission has generally been developed by the larger vertically integrated utilities that typically operate generation and distribution networks. Distribution is handled by a large number of utilities that often only sell electricity. Electricity restructuring has focused primarily on converting the industry to fully compete the sale of electricity production or generation and not the transmission or distribution of electricity. The restructuring of the industry is, in large part, the separating of generation assets from the transmission and distribution assets into separate economic entities in many state efforts. Transmissions and distribution remain price regulated throughout the country based on the cost of service.

# 6.2 Deregulation and Restructuring

The ongoing process of deregulation of wholesale and retail electric markets is changing the structure of the electric power industry. In addition to reorganizing asset management between companies, deregulation is aimed at the functional unbundling of generation, transmission, distribution, and ancillary services the power sector has historically provided to competition in the generation segment of the industry.

Beginning in the 1970s, government policy shifted against traditional regulatory approaches and in favor of deregulation for many important industries, including transportation, communications, and energy, which were all thought to be natural monopolies (prior to 1970) that warranted governmental control of pricing. Some of the primary drivers for deregulation of electric power included the desire for more efficient investment choices, the possibility of lower electric rates, reduced costs of combustion turbine technology that opened the door for more companies to sell power, and complexity of monitoring utilities' cost of service and establishing cost-based rates for various customer classes (see Figure 6-1). The pace of restructuring in the electric power industry slowed significantly in response to market volatility and financial turmoil associated with bankruptcy filings of key energy companies in California. By the end of 2001, restructuring had either been delayed or suspended in eight states that previously enacted legislation or issued regulatory orders for its implementation. Another 18 other states that had seriously explored the possibility of deregulation in 2000 reported no legislative or regulatory activity in 2001 (DOE, EIA, 2003a). Currently, there are 17 states where price deregulation of generation (restructuring) has occurred. Fifteen of these states are in the CAIR region. The effort is more or less at a standstill; however, at the federal level, there are efforts in the form of proposed legislation and proposed Federal Energy Regulatory Commission (FERC) actions aimed at reviving restructuring. For states that have not begun restructuring efforts, it is unclear when and at what pace these efforts will proceed.



# Figure 6-1. Status of State Electricity Industry Restructuring Activities (as of February 2003)

## 6.3 **Pollution and EPA Regulation of Emissions**

The burning of fossil fuels, which generates about 70 percent of our electricity nationwide, results in air emissions of  $SO_2$  and  $NO_x$ , important precursors in the formation of fine particles and ozone ( $NO_x$  only). The power sector is a major contributor of both these pollutants, and reductions of  $SO_2$  and  $NO_x$  emissions are critical to EPA's efforts to bring about attainment with the fine particle and ozone NAAQS through programs like CAIR. In 2003, the power sector accounted for 67 percent of total nationwide  $SO_2$  emissions and 22 percent of total nationwide  $NO_x$  emissions (see Figure 6-2).

Different types of fossil fuel-fired units vary widely in their air emissions levels for  $SO_2$  and  $NO_x$ , particularly when uncontrolled. For coal-fired units,  $NO_x$  emission rates can vary from under 0.05 lbs/mmBtu (for a unit with selective catalytic reduction for  $NO_x$  removal) to over 1 lb/mmBtu for an uncontrolled cyclone boiler.  $NO_x$  emissions from coal-fired power plants are formed during combustion and are a result of both nitrogen in coal and nitrogen in the air.  $SO_2$  emission rates can vary from under 0.1 lbs/mmBtu (for some units with flue gas desulfurization for  $SO_2$  removal) to over 5 lbs/mmBtu for units

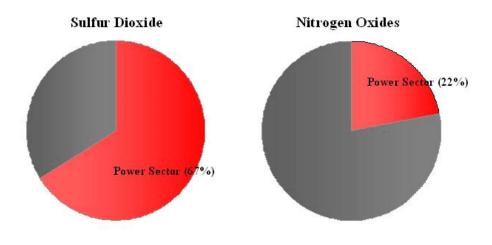


Figure 6-2. Emissions of SO₂ and NO_x from the Power Sector (2003)

burning higher sulfur coal. For an uncontrolled coal plant,  $SO_2$  emissions are directly related to the amount of sulfur in the coal.

Oil and gas-fired units also have a wide range of  $NO_x$  emissions depending on both the plant type and the controls installed. Units with selective catalytic reduction (SCR) can have emission rates under 0.01 lbs/mmBtu, while completely uncontrolled units can have emission rates in excess of 0.5 lbs/mmBtu. Gas-fired units have very little SO₂ emissions.  $NO_x$  emission rates on oil-fired units can range from under 0.1 lbs/mmBtu (for units with new combustion controls) to over 0.6 lbs/mmBtu for units without combustion controls. SO₂ emissions for oil-fired units can range from under 0.1 lbs/mmBtu for units burning low sulfur distillate oil to over 2 lbs/mmBtu for units burning high sulfur residual oil.

# 6.4 Pollution Control Technologies

There are two primary options for reducing SO₂ emissions from coal-burning power plants. Units may switch from higher to lower sulfur coal, or they may use flue gas desulfurization (FGD, commonly referred to as scrubbers). According to data submitted to EPA for compliance with the Title IV Acid Rain Program, the SO₂ emission rates for coal-fired units varied from under 0.4 lbs/mmBtu to over 5 lbs/mmBtu depending on the type of coal combusted.

It is generally easier to switch to a coal within the same rank (e.g., bituminous or sub-bituminous) because these coals will have similar heat contents and other characteristics. Switching completely to sub-bituminous coal (which typically has a lower sulfur content) from bituminous coal is likely to require some modifications to the unit. Limited blending of sub-bituminous coal with bituminous coal can often be done with much more limited modifications.

The two most commonly used scrubber types include wet scrubbers and spray dryers. Wet scrubbers can use a variety of sorbents to capture  $SO_2$  including limestone and magnesium enhanced lime. The choice of sorbent can affect the performance, size, and capital and operating costs of the scrubber. New wet scrubbers typically achieve at least 95 percent  $SO_2$  removal. Spray dryers can achieve over 90 percent removal.

One method of reducing  $NO_x$  emissions is through the use of combustion controls (such as low  $NO_x$  burners and over-fired air). Combustion controls reduce  $NO_x$ , by ensuring that the combustion of coal occurs under conditions under which less formation of  $NO_x$ occurs. Post-combustion controls reduce  $NO_x$  by removing the  $NO_x$  after it has been formed. The most common post-combustion control is SCR. SCR systems inject ammonia (NH3), which combines with the  $NO_x$  in the flue gas, to form nitrogen and water and uses a catalyst to enhance the reaction. These systems can reduce  $NO_x$  by 90 percent and achieve emission rates of around 0.06 lbs/mmBtu. Selective noncatalytic reduction also removes  $NO_x$  by injecting ammonia, but no catalyst is used. These systems can reduce  $NO_x$  by up to 40 percent.

For more detail on the cost and performance assumptions of pollution controls, see the documentation for the Integrated Planning Model (IPM), a dynamic linear programming model that EPA uses to examine air pollution control policies for  $SO_2$  and  $NO_x$  throughout the contiguous United States for the entire power system. Documentation for IPM can be found at www.epa.gov/airmarkets/epa-ipm.

# 6.5 Regulation of the Power Sector

At the federal level, efforts to reduce emissions of  $SO_2$  and  $NO_x$  have been occurring since 1970. Policy makers have recognized the need to address these harmful emissions, and incremental steps have been taken to ensure that the country meets air quality standards. CAIR is the next step towards realizing attainment of the standards.

Federal regulation of  $SO_2$  and  $NO_x$  emissions at power plants began with the 1970 Clean Air Act. The Act required the Agency to develop performance standards for a number of source categories including coal-fired power plants. The first New Source Performance Standards (NSPS) for power plants (subpart D) required new units to limit  $SO_2$  emissions either by using scrubbers or by using low sulfur coal.  $NO_x$  was required to be limited through the use of low  $NO_x$  burners. A new NSPS (subpart Da), promulgated in 1978, tightened the standards for  $SO_2$  requiring scrubbers on all new units.

The 1990 Clean Air Act Amendments (CAAA) placed a number of new requirements on power plants. The Acid Rain Program, established under Title IV of the 1990 CAAA, requires major reductions of SO₂ and NO_x emissions. The SO₂ program sets a permanent cap on the total amount of SO₂ that can be emitted by electric power plants in the contiguous United States at about one-half of the amount of SO₂ these sources emitted in 1980. Using a market-based cap-and-trade mechanism allows flexibility for individual combustion units to select their own methods of compliance. The program uses a more traditional approach to NO_x emission limitations for certain coal-fired electric utility boilers, with the objective of achieving a 2 million ton reduction from projected NO_x emission levels that would have been emitted in 2000 without implementation of Title IV.

The Acid Rain Program comprises two phases for  $SO_2$  and  $NO_x$ . Phase I applied primarily to the largest coal-fired electric generation sources from 1995 through 1999 for  $SO_2$  and from 1996 through 1999 for  $NO_x$ . Phase II for both pollutants began in 2000. For  $SO_2$ , it applies to thousands of combustion units generating electricity nationwide; for  $NO_x$  it generally applies to affected units that burned coal during 1990 through 1995. The Acid Rain Program has led to the installation of a number of scrubbers on existing coal-fired units as well as significant fuel switching to lower sulfur coals. Under the  $NO_x$  provisions of Title IV, most existing coal-fired units were required to install low  $NO_x$  burners.

The CAAA also placed much greater emphasis on control of  $NO_x$  to reduce ozone nonattainment. This has led to the formation of several regional  $NO_x$  trading programs as well as an intrastate  $NO_x$  trading program in Texas. The Ozone Transport Commission (a group of northeast states) created an interstate  $NO_x$  trading program that began in 1999. In 1998, EPA promulgated regulations (the  $NO_x$  SIP Call) that required 21 states in the eastern United States and the District of Columbia to reduce  $NO_x$  emissions that contributed to nonattainment in downwind states using the cap-and-trade approach. This program began in the summer of 2004 and has resulted in the installation of significant amounts of selective catalytic reduction.

In addition to federal programs to reduce emissions of  $SO_2$  and  $NO_x$ , several states have also taken action. Several states, like North Carolina, New York, Connecticut, and Massachusetts, have moved to control these emissions to address nonattainment.

# 6.6 Cap and Trade

The cap-and-trade system under CAIR, which is largely based on the Acid Rain Trading Program and the  $NO_x$  SIP Call, provides the power sector with considerable flexibility in meeting the emission reduction requirements. Cap-and-trade regulation is an extremely efficient tool that allows for environmental goals to be met in the most cost-effective manner, because firms have economic incentives to achieve emissions reductions where they are cheapest. The system allows for various compliance options, with each firm determining what option works best given certain costs, such as fuel costs or costs of pollution controls.

In addition to the pollution control options discussed above, companies can comply with cap-and-trade programs through more efficient use of the generating fleet to take advantage of generating sources that emit less and run more efficiently, commonly referred to as dispatch changes. By shifting generation to these more efficient units, the power sector is reducing the cost of compliance because there is a cost to pollute under a cap. Another option is purchasing additional allowances to cover emissions.

### 6.7 Clean Air Interstate Rule

To address air quality problems and improve public health and the environment, EPA is finalizing CAIR. The final CAIR requires annual  $SO_2$  and  $NO_x$  reductions in 23 States and the District of Columbia, and also requires ozone season  $NO_x$  reductions in 25 States and the District of Columbia. Many of the CAIR States are affected by both the annual  $SO_2$  and  $NO_x$  reduction requirements and the ozone season  $NO_x$  requirements. CAIR allows affected states to adopt a two-phased cap-and-trade program to meet emissions reduction requirements of roughly 73 percent for  $SO_2$  and 61 percent for  $NO_x$  from 2003 levels.

The rule would affect roughly 3,000 fossil fuel-fired units with a nameplate capacity greater than 25 MW. These sources accounted for roughly 89 percent of nationwide  $SO_2$  emissions and 79 percent of nationwide  $NO_x$  emissions in 2003 (see Table 6-4).

Table 6-4. Emissions of SO₂ and NO_x in 2003 and Percentage of Emissions in the CAIR Affected Region (tons)

	SO ₂	NO _x
CAIR Region	9,407,406	3,222,636
Nationwide	10,595,069	4,165,026
CAIR Emissions as % of Nationwide Emissions	89%	79%

Source: EPA.

Note: Region includes states covered for the annual SO₂ and NO_x trading programs (Alabama, District of Columbia, Florida, Georgia, Illinois, Indiana, Iowa, Kentucky, Louisiana, Maryland, Michigan, Minnesota, Mississippi, Missouri, New York, North Carolina, Ohio, Pennsylvania, South Carolina, Tennessee, Texas, Virginia, West Virginia, and Wisconsin).

EPA modeling¹ shows that coal-fired and oil/gas-fired generation will continue to play an important part of the electricity generating portfolio in the United States. Electricity demand is anticipated to grow by 1.6 percent a year, and total electricity demand is projected to be 4,198 billion kWh by 2010. Table 6-5 shows current electricity generation and projected levels in 2010 and 2015 using EPA modeling.

# Table 6-5. Current Electricity Net Generation and EPA Projections for 2010 and 2015(Billion kWh)

	2003	2010	2015
Coal	1,970	2,198	2,242
Oil/Gas	758	777	1,026
Other	1,119	1,223	1,235
Total	3,848	4,198	4,503

Source: 2003 data is from EIA. Projections are from the Integrated Planning Model run by EPA.

¹EPA uses the IPM to make power-sector forecasts about emissions, costs, and other key factors of the power sector. Industry projections presented here are from EPA's base case scenario. For more information about IPM, see http://www.epa.gov/airmarkets/epa-ipm/index.html.

# 6.8 Price Elasticity of Electricity

Electricity performs a vital and high-value function in the economy; as a result, electricity consumers are generally unable or unwilling to alter consumption as the price increases. Demand for electricity, especially in the short run, is not very sensitive to changes in prices and is considered relatively price inelastic although some demand reduction does occur. With that in mind, EPA modeling does not incorporate a "demand response" to any increases in electricity prices because of the reasons mentioned. Electricity demand is considered to be constant in EPA modeling applications and the reduction in production costs that would result from lower demand is not considered. This leads to some overstatement in the private compliance costs that EPA estimates.

### **CHAPTER 7**

## COST, ECONOMIC, AND ENERGY IMPACTS

This chapter reports the cost, economic, and energy impact analysis performed for CAIR. EPA used the IPM, developed by ICF Consulting, to conduct its analysis. IPM is a dynamic linear programming model that can be used to examine air pollution control policies for  $SO_2$  and  $NO_x$  throughout the contiguous United States for the entire power system. Documentation for IPM can be found at www.epa.gov/airmarkets/epa-ipm.

### 7.1 Modeling Background

The analysis presented here covers the electric power sector, a major source of  $SO_2$  and  $NO_x$  emissions nationwide and the industry that EPA assumes that States will control in setting State emissions reduction requirements. EPA has also assumed that States implement those reductions through a cap-and-trade program. For  $SO_2$  and  $NO_x$ , EPA modeled an annual, two-phased control strategy for 26 eastern States and the District of Columbia (see Figure 7-1). For  $NO_x$ , separate ozone season caps were applied to Connecticut and Massachusetts. See Table 7-1 for total annual emissions caps under CAIR.



Figure 7-1. CAIR Modeled Region

	2010–2014 ('09-'14 for NO _x )	2015–Thereafter
SO ₂	3.6	2.5
NO _x	1.5	1.3

 Table 7-1. CAIR Annual Emissions Caps (Million Tons)

The final CAIR requires annual  $SO_2$  and  $NO_x$  reductions in 23 States and the District of Columbia, and also requires ozone season  $NO_x$  reductions in 25 States and the District of Columbia. Many of the CAIR States are affected by both the annual  $SO_2$  and  $NO_x$  reduction requirements and the ozone season (May-September)  $NO_x$  requirements. Using IPM, EPA modeled the cost and emissions impacts of CAIR. For further discussion about the scope and requirements of CAIR, see the Final CAIR preamble.

EPA initially conducted IPM modeling for today's final action using a control strategy that is similar, but not identical to, the final CAIR requirements. The control strategy that EPA initially modeled included three additional States (Arkansas, Delaware and New Jersey) within the region and required these States to make annual SO₂ and NO_x reductions. However, these three States are not required to make annual reductions under the final CAIR. In the "Proposed Rules" section of today's Federal Register publication, EPA is publishing a proposal to include Delaware and New Jersey in the CAIR region for annual SO₂ and NO_x reductions. This RIA is to serve as EPA's analytical assessment of both today's Final CAIR and the proposed rule for incorporating Delaware and New Jersey into the annual SO₂ and NO_x requirements of CAIR. The addition of Arkansas, Delaware, and New Jersey brought the total number of affected States for annual SO₂ and NO_x to 26 plus the District of Columbia for the initial model run. Arkansas will not be included in the annual SO₂ and NO_x requirements either as part of today's Final CAIR or the "Proposed Rule," but is included for the ozone season CAIR requirement as part of today's Final Rule. The initial model run also included individual State ozone season NO_x caps for Connecticut and Massachusetts, and did not include ozone season NO_x caps for any other CAIR States.

The Agency conducted revised final IPM modeling that reflects the final CAIR control strategy. The final IPM modeling includes regionwide annual  $SO_2$  and  $NO_x$  caps on the 23 States and the District of Columbia for States required to make annual reductions, and includes a regionwide ozone season  $NO_x$  cap on the 25 States and the District of Columbia required to make ozone season reductions. EPA modeled the final CAIR  $NO_x$  strategy as an annual  $NO_x$  cap with a nested, separate ozone season  $NO_x$  cap.

In this chapter of the RIA, the projected CAIR costs and emissions are derived from the IPM run reflecting CAIR with Arkansas, Delaware, and New Jersey included for the annual requirements, and without a separate ozone season  $NO_x$  cap for ozone season States. However, where IPM results differ significantly between the scenario with Arkansas, Delaware, and New Jersey, and the scenario with these three States included for  $NO_x$  summer season only, EPA has highlighted these differences and included them in this chapter as well. The air quality and benefits analyses done in support of CAIR are based on emission projections from the initial IPM run with Arkansas, Delaware, and New Jersey included for annual SO₂ and  $NO_x$ .

EPA believes that the differences between the initial IPM run and the final IPM run have very little impact on projected control costs, emissions, and other impacts. Modeling the CAIR region without Arkansas, Delaware, and New Jersey do not change the results presented here in any significant way, and in any event, this chapter generally reflects the geographic scope of the CAIR program as EPA intends it to be ultimately. IPM output files for the model runs used in CAIR analyses are available in the CAIR docket.

CAIR was designed to achieve significant emissions reductions in a highly cost-effective manner to reduce the transport of fine particles that have been found to contribute to nonattainment. EPA analysis has found that the most efficient method to achieve the emissions reduction targets is through a cap-and-trade system on the power sector that States have the option of adopting. The power sector accounted for 67 percent of nationwide SO₂ emissions and 22 percent of nationwide NO_x emissions in 2002. States, in fact, can choose not to participate in the optional cap-and-trade program and can choose to obtain equivalent emissions reductions from other sectors. However, EPA believes that a region-wide cap-and-trade system for the power sector is the best approach for reducing emissions. The modeling done with IPM assumes a region-wide cap and trade system on the power sector for the States covered. However, EPA recognizes that States may choose to cover other sources and may use a different approach for reducing emissions.

IPM has been used for evaluating the economic and emission impacts of environmental policies for over a decade. The model's base case incorporates title IV of the Clean Air Act (the Acid Rain Program), the NO_x SIP Call, various New Source Review (NSR) settlements, and several State rules affecting emissions of SO₂ and NO_x that were finalized prior to April of 2004. The NSR settlements include agreements between EPA and Southern Indiana Gas and Electric Company (Vectren), Public Service Enterprise Group, Tampa Electric Company, We Energies (WEPCO), Virginia Electric & Power Company (Dominion), and Santee Cooper. IPM also includes various current and future State programs in Connecticut, Illinois, Maine, Massachusetts, Minnesota, New Hampshire, North Carolina, New York, Oregon, Texas, and

Wisconsin. IPM includes State rules that have been finalized and/or approved by a State's legislature or environmental agency. The base case is used to provide a reference point to compare environmental policies and assess their impacts and does not reflect a future scenario that EPA predicts will occur.

The economic modeling presented in this chapter has been developed for specific analyses of the power sector. Thus, the model has been designed to reflect the industry as accurately as possible. As a result, EPA has used discount rates in IPM that are appropriate for the various types of investments and other costs that the power sector incurs. The discount rates used in IPM may differ from discount rates used in other EPA analyses done for CAIR, particularly the discount rates used in the benefits analysis that are assumed to be social discount rates. EPA uses the best available information from utilities, financial institutions, debt rating agencies, and government statistics as the basis for the discount rates used for power sector modeling. These discount rates have undergone review by the power sector and the Energy Information Administration. EPA's discount rate approach has not been challenged in court.

EPA's modeling is based on its best judgment for various input assumptions that are uncertain, particularly assumptions for future fuel prices and electricity demand growth. To some degree, EPA addresses the uncertainty surrounding these two assumptions through its sensitivity analysis, which is discussed in Appendix D.

More detail on IPM can be found in the model documentation, which provides additional information on the assumptions discussed here as well as all other assumptions and inputs to the model (www.epa.gov/airmarkets/epa-ipm).

### 7.2 Projected SO₂ and NO_x Emissions and Reductions

Because of the existence of a bank of allowances under the title IV Acid Rain Program that sources will be allowed to use under the requirements of CAIR, emissions of SO₂ in 2010 and 2015 will be higher than the caps that are required for CAIR. Table 7-2 provides projected emissions levels.

As shown in Figure 7-2, the results of EPA modeling of CAIR show that substantial  $SO_2$  emissions reductions occur in the Midwest and Mid-Atlantic regions of the country. Significant  $NO_x$  emissions reductions occur across the CAIR region (see Figure 7-3), and with CAIR, ozone season  $NO_x$  emissions reductions are lower than they would have been with the  $NO_x$  SIP Call (base case) (see Figure 7-4). For  $NO_x$ , the annual CAIR cap achieves greater emission reductions during the ozone season than the  $NO_x$  SIP Call summer requirement.

Table 7-2. Projected Emissions of  $SO_2$  and  $NO_x$  with the Base Case^a (No Further Controls) and with CAIR (Million Tons)

			2010			2015			2020	
	Coverage	Base Case	CAIR	Emission Reductions	Base Case	CAIR	Emission Reductions	Base Case	CAIR	Emission Reductions
SO ₂	Nationwide	9.7	6.1	3.6	8.9	4.9	4.0	8.6	4.2	4.5
(annual)	CAIR Region	8.8	5.2	3.6	8.0	4.1	4.0	7.9	3.4	4.5
NO _x	Nationwide	3.6	2.4	1.2	3.7	2.1	1.5	3.7	2.1	1.6
(annual)	CAIR Region	2.8	1.5	1.3	2.8	1.3	1.5	2.9	1.3	1.6
NO _x	Nationwide	1.2	1.0	0.2	1.2	0.9	0.3	1.2	1.0	0.3
(summer)	CAIR Region	0.8	0.7	0.2	0.8	0.6	0.3	0.8	0.6	0.3

Note: Numbers may not add due to rounding. The emissions data presented here are EPA modeling results and the CAIR region includes States modeled for the annual  $SO_2$  and  $NO_x$  requirements. "Summer" is from May 1–September 30, which is the ozone season.

^a Base case includes title IV Acid Rain Program, NO_x SIP Call, and State rules finalized before March 2004. Source: Integrated Planning Model run by EPA.

# 7.3 **Projected Costs**

For the modeled region, EPA projects that the annual incremental costs of CAIR are \$2.4 billion in 2010 and \$3.6 billion in 2015 (see Table 7-3). In 2020, the annual costs are \$4.4 billion. The cost of electricity generation represents roughly one-third to one-half of total electricity costs, with transmission and distribution costs representing the remaining portion. A better impact measure is the impact on electricity pricing, which is shown in a later table. The marginal costs of CAIR for SO₂ and NO_x can also be found in Table 7-3.

### 7.4 Projected Control Technology Retrofits

CAIR is projected to result in the installation of an additional 64 GW of flue gas desulfurization (scrubbers) on existing coal-fired generation capacity for SO₂ control and an additional 34 GW of selective catalytic reduction technology (SCR) on existing coal-fired generation capacity for NO_x control by 2015 (see Table 7-4). The first phase of CAIR will result in 37 GW of additional scrubbers and 14 GW of SCR by 2010. Much of the NO_x reductions

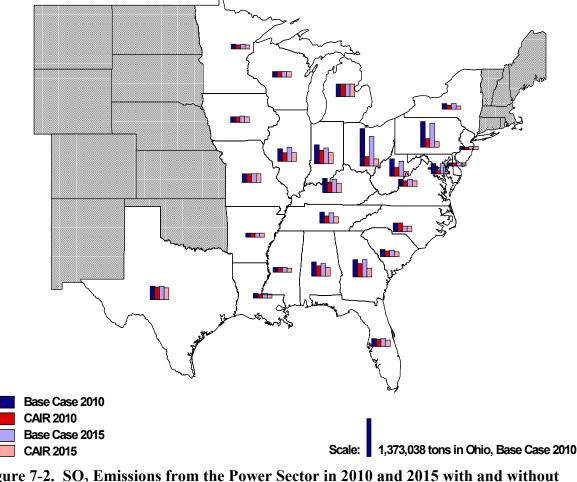
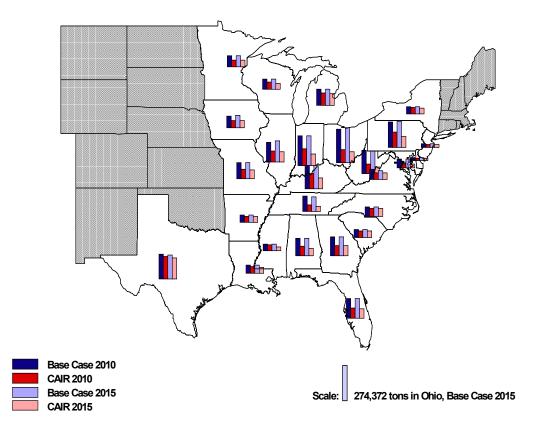


Figure 7-2.  $SO_2$  Emissions from the Power Sector in 2010 and 2015 with and without CAIR

Source: Integrated Planning Model run by EPA.

Note: Arkansas, Delaware, and New Jersey are not included in the annual  $SO_2$  and  $NO_x$  requirements of the Final CAIR. These States are included in the ozone season requirement only. Modeling presented here did not include an ozone season requirement and covered a region that is inclusive of these three States for the annual requirements. Please see earlier modeling background discussion for more detail.



# Figure 7-3. NO_x Emissions from the Power Sector in 2010 and 2015 With and Without CAIR.

Source: Integrated Planning Model run by EPA.

Note: Arkansas, Delaware, and New Jersey are not included in the annual  $SO_2$  and  $NO_x$  requirement of the Final CAIR. These States are included in the ozone season requirement only. Modeling presented here did not include an ozone season requirement and covered a region that is inclusive of these three States for the annual requirements. Please see earlier modeling background discussion for more detail.

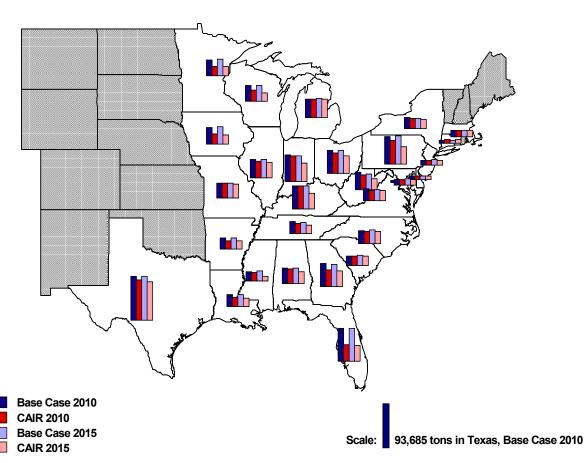


Figure 7-4. Ozone Season  $\mathrm{NO}_{\mathrm{X}}$  Emissions from the Power Sector in 2010 and 2015 with and without CAIR

Source: Integrated Planning Model run by EPA.

Note: Arkansas, Delaware, and New Jersey are not included in the annual  $SO_2$  and  $NO_x$  requirements of the Final CAIR. These States are included in the ozone season requirements only. Modeling presented here did not included an ozone season requirement and covered a region that is inclusive of these three States for the annual requirements. Please see earlier modeling background discussion for more detail.

# Table 7-3. Annualized Regional Cost of CAIR and Marginal Cost of SO₂ and NO_x Reductions with CAIR (\$1999)

		2010	2015	2020
Annualized Cost (billions)		\$2.4	\$2.4 \$3.6	
Manainal Cast (@/tan)	SO ₂	\$700	\$1,000	\$1,400
Marginal Cost (\$/ton)	NO _x	\$1,300	\$1,600	\$1,600

Note: Numbers rounded to the nearest hundred million for annualized cost and nearest hundred for marginal cost. Source: Integrated Planning Model run by EPA.

# Table 7-4. Pollution Controls by Technology with the Base Case (No Further Controls) and with CAIR (GW)

	Base Case Total (Cumulative)		Incremental with CAIR				tal with CA Cumulativ		
Technology	2010	2015	2020	2010	2015	2020	2010	2015	2020
Scrubbers	110	116	117	37	64	82	147	180	199
SCR	111	119	121	14	34	33	126	152	154

Note: Numbers may not add due to rounding. Base case retrofits include existing scrubbers and SCR as well as additional retrofits for the Title IV Acid Rain Program, the NO_x SIP call, NSR settlements, and various State rules.

Source: Integrated Planning Model run by EPA.

achieved in the first phase of the rule can be attributed to the large pool of existing SCR that are used during the ozone season in the  $NO_x$  SIP call region that, for relatively little cost, run the SCRs year-round. A small number of coal-fired units also install selective noncatalytic reduction technology (SNCR) for  $NO_x$  control under CAIR. Emission reductions are achieved through a combination of compliance options, such as additional pollution control installations, generation shifts towards more efficient electricity producing units, and fuel and coal switching.

### 7.5 Projected Generation Mix

Table 7-5 and Figure 7-5 show the generation mix with CAIR. Coal-fired generation and natural gas-fired generation are projected to remain relatively unchanged because of the phasedin nature of CAIR, which allows industry the appropriate amount of time to install the necessary pollution controls.

# Table 7-5. Generation Mix with the Base Case (No Further Controls) and with CAIR (Thousand GWhs)

		2010			2015			2020		
Generating Fuel Use	2003	Base Case	CAIR	Change from Base Case	Base Case	CAIR	Change from Base Case	Base Case	CAIR Percent Change	Change from Base Case
Coal	1,970	2,198	2,163	-1.6%	2,242	2,195	-2.1%	2,410	2,381	-1.2%
Oil/Natural Gas	758	777	809	4.1%	1,026	1,072	4.5%	1,221	1,250	2.3%
Other	1,120	1,223	1,218	-0.4%	1,235	1,233	-0.2%	1,218	1,217	-0.1%
Total	3,848	4,198	4,190	-0.2%	4,503	4,499	-0.1%	4,850	4,847	-0.1%

Note: Numbers may not add due to rounding.

Source: 2003 data are from EIA and projections are from the Integrated Planning Model run by EPA.

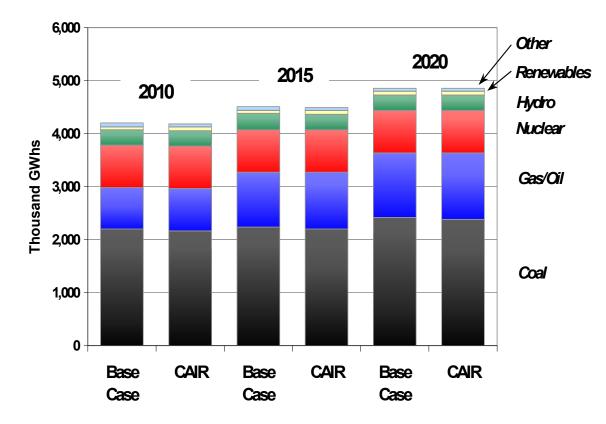


Figure 7-5. Generation Mix with and without CAIR

Source: Integrated Planning Model run by EPA.

Relative to the base case, about 5.3 GW of coal-fired capacity is projected to be uneconomic to maintain (about 1.7 percent of all coal-fired capacity and 0.5 percent of all generating capacity), and about 1 GW of coal-fired capacity is projected to repower to natural gas or Integrated Gasification Combined Cycle (IGCC). Uneconomic units, for the most part, are small and infrequently used generating units that are dispersed throughout the CAIR region. In practice, units projected to be uneconomic to maintain may be "mothballed," retired, or kept in service to ensure transmission reliability in certain parts of the grid. EPA modeling is unable to distinguish between these potential outcomes. IPM can only predict that specific generating units are uneconomic to maintain, based on their fuel, operating and fixed costs, and whether they are needed to meet both demand and reliability reserve requirements. "Repowering" converts units to combined-cycle natural gas or IGCC.

# 7.6 Projected Capacity Additions

In addition, EPA projects that future growth in electric demand will be met with a combination of new natural gas- and coal-fired capacity (see Table 7-6).

	Current	Base Case	CAIR
Pulverized Coal	305	318	314
IGCC	0.6	8	9
Oil/Gas	395	467	469

### Table 7-6. Total Coal and Natural Oil/Gas-Fired Capacity by 2020 (GW)

Source: Current data are from EPA's NEEDS 2004. Projections are from the Integrated Planning Model run by EPA.

### 7.7 Projected Coal Production for the Electric Power Sector

Coal production for electricity generation is expected to increase relative to current levels, with or without CAIR (see Table 7-7 and Figure 7-6). The reductions in emissions from the power sector will be met through the installation of pollution controls for  $SO_2$  and  $NO_x$  removal. The pollution controls can achieve up to a 95 percent  $SO_2$  removal rate, which allows industry to rely more heavily on local bituminous coal in the eastern and central parts of the country that has a higher sulfur content and is less expensive to transport than western subbituminous coal.

# 7.8 Projected Retail Electricity Prices

Retail electricity prices for the CAIR region are projected to increase a small amount with CAIR (see Table 7-8 and Figure 7-7). The cap-and-trade approach allows industry to meet

 Table 7-7. Coal Production for the Electric Power Sector with the Base Case (No Further Controls) and with CAIR (Million Tons)

				Base Case		CAIR				
Supply Area	2000	2003	2010	2015	2020	2010	2015	2020		
Appalachia	299	275	325	315	301	306	310	331		
Interior	131	135	161	162	173	164	193	219		
West	475	526	603	631	714	607	579	607		
National	905	936	1,089	1,109	1,188	1,077	1,082	1,156		

Source: 2000 and 2003 data are derived from EIA data. All projections are from the Integrated Planning Model run by EPA.

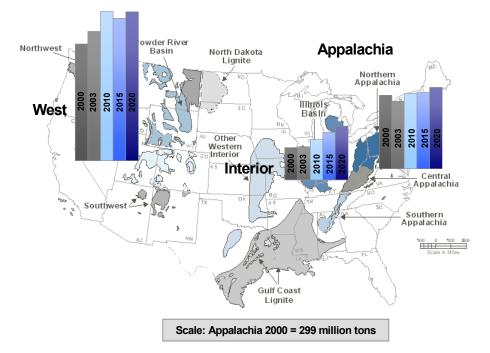


Figure 7-6. Current Coal Production Levels and Projected Production with CAIR

Source: Integrated Planning Model run by EPA.

 Table 7-8. Projected Regional Retail Electricity Prices with the Base Case (No Further Controls) and with CAIR (Mills/kWh)

Year	Base Case	CAIR	Percent Change
2010	58	59	2.0%
2015	61	62	2.7%
2020	61	62	1.8%

Source: EPA's Retail Electricity Price Model.

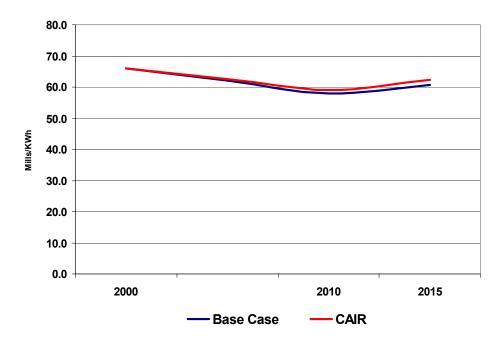


Figure 7-7. Regional Electricity Prices with and without CAIR

Source: Integrated Planning Model run by EPA.

the requirements of CAIR in the most cost-effective manner, thereby minimizing the costs passed on to consumers. Regional retail electricity prices are projected to be 2 to 3 percent higher with CAIR. Retail electricity prices by NERC region are provided in Table 7-9 (Figure 7-8). These results show small increases in retail prices for the NERC regions in the eastern part of the country. By 2020, CAIR region retail electricity prices are projected to be roughly 1.8 percent higher with CAIR (Table 7-8 and Figure 7-7).

Power		Base Case			CAIR			Percentage Change			
Region	Primary States Included	2000	2010	2015	2020	2010	2015	2020	2010	2015	2020
ECAR (1)	OH, MI, IN, KY, WV, PA	57.4	51.7	55.2	56.1	53.8	58.5	58.0	4.0%	5.9%	3.4%
ERCOT (2)	TX	65.1	57.9	64.4	62.6	59.3	64.6	63.3	2.5%	0.2%	1.2%
MAAC (3)	PA, NJ, MD, DC, DE	80.4	59.3	69.4	72.2	61.2	71.7	72.8	3.2%	3.4%	0.8%
MAIN (4)	IL, MO, WI	61.2	52.6	57.8	61.0	54.0	60.3	62.0	2.6%	4.3%	1.7%
MAPP (5)	MN, IA, SD, ND, NE	57.4	52.8	49.3	47.6	52.9	49.6	48.0	0.2%	0.7%	0.8%
NY (6)	NY	104.3	82.8	87.9	88.1	83.3	88.8	88.4	0.5%	1.0%	0.3%
NE (7)	VT, NH, ME, MA, CT, RI	89.9	77.4	83.9	82.8	77.5	84.7	83.0	0.1%	1.0%	0.2%
FRCC (8)	FL	67.9	71.2	71.3	69.5	71.7	72.3	70.5	0.8%	1.3%	1.5%
STV (9)	VA, NC, SC, GA, AL, MS,	59.3	56.2	55.1	55.3	57.0	56.2	56.6	1.5%	2.1%	2.3%
	TN, AR, LA										
SPP (10)	KS, OK, MO	59.3	54.2	57.0	56.7	54.6	57.5	57.0	0.7%	0.9%	0.6%
Regionwide		66.0	58.0	60.8	61.0	59.2	62.4	62.1	2.0%	2.7%	1.8%

Table 7-9. Retail Electricity Prices by NERC Region with the Base Case (No Further Controls) and with CAIR (Mills/kWh)

Source: EPA's Retail Electricity Price Model. 2000 prices are from EIA's AEO 2003.

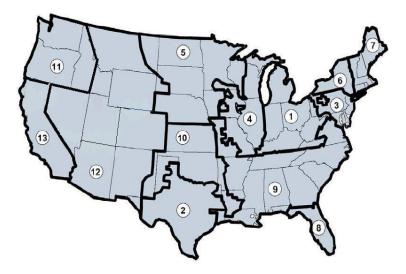


Figure 7-8. NERC Power Regions

#### 7.9 **Projected Fuel Price Impacts**

The impacts of CAIR on coal prices and natural gas prices before shipment are shown below (Table 7-10).

Table 7-10. Henry Hub Natural Gas Prices and Average Delivered Coal Prices with the
Base Case (No Further Controls) and with CAIR (\$1999)

		Base Case			CAIR			Percentage Change		
Fuel	2000	2010	2015	2020	2010	2015	2020	2010	2015	2020
Natural Gas	4.15	3.20	3.25	3.16	3.25	3.30	3.20	1.6%	1.5%	1.3%
Coal	1.25	1.05	1.01	0.96	1.05	0.98	0.93	0.0%	-3.0%	-3.1%

Source: Integrated Planning Model run by EPA. 2000 natural gas data are from Platts GASdat. 2000 coal prices are from EIA.

Note: Coal price changes largely result from changes in the mix of coal types used. Delivered coal prices vary widely, but large changes in the cost of each type of coal are not projected.

### 7.10 Key Differences in EPA Model Runs for Final CAIR Modeling

As previously stated, the emissions, cost, air quality, and benefits analyses done for the final CAIR are from a modeling scenario that requires annual  $SO_2$  and  $NO_x$  reductions in 26 States and the District of Columbia and ozone season  $NO_x$  requirements in Connecticut and Massachusetts (See Figure 7-1). This modeling differs from today's final CAIR, in that Arkansas, Delaware, and New Jersey are not included in the annual  $SO_2$  and  $NO_x$  requirements, and various States are required to meet an ozone season  $NO_x$  requirement (See Figure 7-9). Modeling was done based upon the Final CAIR region, and in large part, results under this scenario are not significantly different in scope or magnitude.

Coal production, minemouth coal and wellhead natural gas prices, generation and generating capacity, and electricity prices do not differ significantly between the scenarios (all less than a 1 percent difference in modeling results). Nationwide emissions of SO₂ and NO_x are roughly 1 to 3 percent higher when Arkansas, Delaware, and New Jersey are not included in the annual programs. The only other notable difference in the modeling results is the difference in retrofits; retrofits of SCR and FGD are roughly 2-4 percent lower without these three States. All IPM runs done in support of CAIR and used as part of the final CAIR package are in the final CAIR Docket and can be found on EPA's website: (http://www.epa.gov/airmarkets/epa-ipm/iaqr.html). A complete list of IPM runs can be found in Appendix D of this RIA.

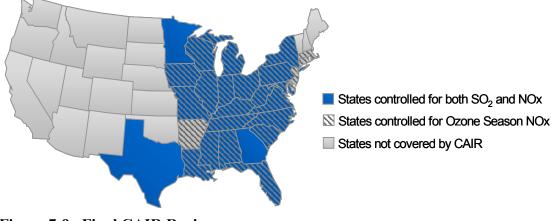


Figure 7-9. Final CAIR Region

Note: Delaware and New Jersey are not included in the Final CAIR for the annual  $SO_2$  and  $NO_x$  requirements. However, EPA intends on incorporating these two States in the annual CAIR program through a separate rulemaking. See earlier discussion for more detail.

In addition, EPA did an analysis to determine the incremental impact of including Delaware and New Jersey in the annual  $SO_2$  and  $NO_x$  requirements from inclusion in the ozone season only  $NO_x$  requirement. Inclusion of these two States leads to roughly an additional fifty thousand tons of  $SO_2$  reduction and ten thousand tons of  $NO_x$  reduction in 2015. The incremental cost of including these two States is estimated to be roughly \$40 million in 2015. Please see "Proposed Rules" section of today's Federal Register publication for more detail on the inclusion of Delaware and New Jersey for the annual CAIR requirements.

### 7.11 Projected Primary PM Emissions from Power Plants

IPM does not project primary PM emissions from power plants. These emissions are projected using a combination of IPM outputs and emission factors. Separate methodologies are used to project filterable PM emissions and condensible PM emissions. The sum is the total projected primary PM emissions.

For filterable PM emissions, emission factors were developed for each unit based on historical information regarding installed emissions controls and types of fuel burned. This methodology tends to underpredict reductions in filterable PM emissions between the base case and the control case because no changes are assumed in the emission factors even if a unit is projected to install a control such as an FGD, which could lead to a decrease in filterable PM emissions.

For condensible PM emissions, emission factors were changed between the base case and the control case to reflect  $SO_2$  controls projected to be installed in the control case. Although

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EPA used the best emission factors available for its analysis, these emission factors did not account for the potential changes in condensible PM emissions due to the installations of SCRs. The formation of additional condensible PM (in the form of  $SO_3$  and  $H_2SO_4$ ) in units with SCRs depends on a number of factors, including coal sulfur content, combustion conditions and characteristics of the catalyst used in the SCR, and is likely to vary widely from unit to unit. SCRs are generally designed and operated so that they minimize increases in condensible PM. This limitation leads to an overprediction of reductions in condensible PM emissions for units with SCRs. For a more complete description of the methodologies used to project PM emissions see "Document for the 2001 Electrical Generating Unit (EGU) Trends Procedures Report," Section 4.2, September 25, 2003.

# 7.12 Limitations of Analysis

EPA's modeling is based on its best judgment for various input assumptions that are uncertain. Assumptions for future fuel prices and electricity demand growth deserve particular attention because of the importance of these two key model inputs to the power sector. To some degree, EPA addresses the uncertainty surrounding these two assumptions through its sensitivity analysis, which is discussed in Appendix D. As a general matter, the Agency selects the best available information from available engineering studies of air pollution controls and has set up what it believes is the most reasonable modeling framework for analyzing the cost, emission changes, and other impacts of regulatory controls.

The annualized cost estimates of the private compliance costs that are provided in this analysis are meant to show the increase in production (engineering) costs of CAIR to the power sector. In simple terms, the private compliance costs that are presented are the annual increase in revenues required for the industry to be as well off after CAIR is implemented as before. To estimate these annualized costs, EPA uses a conventional and widely-accepted approach that is commonplace in economic analysis of power sector costs for estimating engineering costs in annual terms. For estimating annualized costs, EPA has applied a capital recovery factor (CRF) multiplier to capital investments and added that to the annual incremental operating expenses. The CRF is derived from estimates of the cost of capital (private discount rate), the amount of insurance coverage required, local property taxes, and the life of capital. The private compliance costs presented earlier are EPA's best estimate of the direct private compliance costs of CAIR.

The annualization factor used for pure social cost calculations (for annualized costs) normally includes the life of capital and the social discount rate. For purposes of benefit-cost analysis of this rule, EPA has calculated the annualized social costs using the discount rates from the benefits analysis for CAIR (3 percent and 7 percent and a 30 year life of capital. The cost of added insurance necessary because of CAIR was included in the calculations, but local taxes

were not included because they are considered to be transfer payments, and not a social cost). Using these discount rates, the social costs of CAIR are \$1.91 billion in 2010 and \$2.56 billion in 2015 using a discount rate of 3 percent, and \$2.14 billion in 2010 and \$3.07 billion in 2015 using a discount rate of 7 percent.

The annualized regional cost of CAIR, as quantified here, is EPA's best assessment of the cost of implementing CAIR, assuming that States adopt the model cap and trade program. These costs are generated from rigorous economic modeling of changes in the power sector due to CAIR. This type of analysis using IPM has undergone peer review and federal courts have upheld regulations covering the power sector that have relied on IPM's cost analysis.

The direct private compliance cost includes, but is not limited to, capital investments in pollution controls, operating expenses of the pollution controls, investments in new generating sources, and additional fuel expenditures. EPA believes that the cost assumptions used for CAIR reflect, as closely as possible, the best information available to the Agency today. The relatively small cost associated with monitoring emissions, reporting, and record keeping for affected sources is not included in these annualized cost estimates, but EPA has done a separate analysis and estimated the cost to be less than \$42 million (see Section X. B. Paperwork Reduction Act).

Furthermore, there are some unquantified costs that EPA wants to identify as limits to its analysis. These costs include the costs of federal and State administration of the program, which we believe are modest given our experience with the Acid Rain Program and the NO_x Budget Trading Program and likely to be less than the alternative of States developing approvable SIPs, securing EPA approval of those SIPs, and Federal/State enforcement to deal with the air pollution transport problem that CAIR addresses. There also may be unquantified costs of transitioning to CAIR, such as the costs associated with the retirement of smaller or less efficient electricity generating units, and employment shifts as workers are retrained at the same company or re-employed elsewhere in the economy. There are certain relatively small permitting costs associated with Title IV that new program entrants face (we believe there are far less than 1,000 new entrants who may require one day of additional work for trading permits). In a separate analysis explained later in this RIA, the EPA estimates the indirect costs and impacts of higher electricity prices on the entire economy (see Regulatory Impact Analysis for the Final Clean Air Interstate Rule, Appendix E [March 2005]).

Cost estimates for CAIR are based on results from ICF's Integrated Planning Model. The model minimizes the costs of producing electricity (including abatement costs) while meeting load demand and other constraints (full documentation for IPM can be found at www.epa.gov/airmarkets/epa-ipm). The structure of the model assumes that the electric utility industry will be able to meet the environmental emission caps at least cost. Montgomery (1972) has shown that this least cost solution corresponds to the equilibrium of an emission permit system. See also Atkinson and Tietenburg (1982), Krupnick et al. (1980), and McGartland and Oates (1985). However, to the extent that transaction and/or search costs, combined with institutional barriers, restrict the ability of utilities to exhaust all the gains from emissions trading, costs are underestimated by the model. Utilities in the IPM model also have "perfect foresight." To the extent that utilities misjudge future conditions affecting the economics of pollution control, costs may be understated as well.

From another vantage point, this modeling analysis does not take into account the potential for advancements in the capabilities of pollution control technologies for SO₂ and NO_x removal as well as reductions in their costs over time. Market-based cap and trade regulation serves to promote innovation and the development of new and cheaper technologies. As an example, recent cost estimates of the Acid Rain SO₂ trading program by Resources for the Future (RFF) and MIT's Center for Energy and Environmental Policy Research (CEEPR) have been as much as 83 percent lower than originally projected by the EPA (see Carlson et al., 2000; Ellerman, 2003). It is important to note that the original analysis for the Acid Rain Program done by EPA also relied on an optimization model like IPM. Ex ante, EPA cost estimates of roughly \$2.7 to \$6.2 billion¹ in 1989 were an overestimate of the costs of the program in part because of the limitation of economic modeling to predict technological improvement of pollution controls and other compliance options such as fuel switching. Ex post estimates of the annual cost of the Acid Rain SO₂ trading program range from \$1.0 to \$1.4 billion. Harrington et al. have examined cost analyses of EPA programs and found a tendency for predicted costs to overstate actual implementation costs in market-based programs (Harrington, Morgenstern, and Nelson, 2000). In recognition of this, EPA's mobile source program uses adjusted engineering cost estimates of pollution control equipment and installation costs to account for this fact, which EPA has not done in this case.²

It is also important to note that the capital cost assumptions for scrubbers used in EPA modeling applications are highly conservative. These are a substantial part of the compliance costs. Data available from recent published sources show the reported FGD costs from recent installations to be below the levels projected by IPM.³ In addition, EPA also conducted a survey

¹ 2010 Phase II cost estimate in \$1995.

² See recent regulatory impact analysis for the Tier 2 Regulations for passenger vehicles (1999) and Heavy-Duty Diesel Vehicle Rules (2000). There is also evidence that scrubber costs will decrease in the future because of the learning-by-doing phenomenon, as more scrubbers are installed (see Manson, Nelson, and Neumann, 2002).

³ There is also evidence that scrubber costs will decrease in the future because of the learning-by-doing phenomenon, as more scrubbers are installed (see Manson, Nelson, and Neumann, 2002).

of recent FGD installations and compared the costs of these installations to the costs used in IPM. This survey included small, mid-size, and large units. Examples of the comparison of these referenced published data with the FGD capital cost estimates obtained from IPM are provided in the Final CAIR docket.

EPA's latest update of IPM incorporates State rules or regulations adopted before March 2004 and various NSR settlements. Documentation for IPM can be found at www.epa.gov/airmarkets/epa-ipm. Any State or settlement action since that time has not been accounted for in our analysis in this chapter.

As configured in this application, the IPM model does not take into account demand response (i.e., consumer reaction to electricity prices). The increased retail electricity prices shown in Tables 7-8 and 7-9 would prompt end users to curtail (to some extent) their use of electricity and encourage them to use substitutes.⁴ The response would lessen the demand for electricity, resulting in electricity price increases slightly lower than IPM predicts, which would also reduce generation and emissions. Because of demand response, certain unquantified negative costs (i.e., savings) result from the reduced resource costs of producing less electricity because of the lower quantity demanded. To some degree, these saved resource costs will offset the additional costs of pollution controls and fuel switching that we would anticipate with CAIR. Although the reduction in electricity use is likely to be small, the cost savings from such a large industry (\$250 billion in revenues in 2003) is likely to be substantial. EIA analysis examining multi-pollutant legislation under consideration in 2003 indicates that the annualized costs of CAIR may be overstated substantially by not considering demand response, depending on the magnitude and coverage of the price increases.⁵

Recent research suggests that the total social costs of a new regulation may be affected by interactions between the new regulation and pre-existing distortions in the economy, such as taxes. In particular, if cost increases due to a regulation are reflected in a general increase in the price level, the real wage received by workers may be reduced, leading to a small fall in the total amount of labor supplied. This "tax interaction effect" may result in an increase in deadweight loss in the labor market and an increase in total social costs. Although there is a good case for the existence of the tax interaction effect, recent research also argues for caution in making prior assumptions about its magnitude. However, there are currently no government-wide economic

⁴ The degree of substitution/curtailment depends on the price elasticity of demand for electricity.

⁵ See "Analysis of S. 485, the Clear Skies Act of 2003, and S. 843, the Clean Air Planning Act of 2003." Energy Information Administration. September, 2003. EIA modeling indicated that the Clear Skies Act of 2003 (a nationwide cap and trade program for SO₂, NO_x, and mercury), demand response could lower present value costs by as much as 47% below what it would have been without an emission constraint similar to CAIR.

analytical guidelines which discuss the tax interaction effect and its potential relevance for estimation of federal program costs and benefits. The limited empirical data available to support quantification of any such effect leads to this qualitative identification of the costs.

On balance, after consideration of various unquantified costs (and savings that are possible), EPA believes that the annual private compliance costs that we have estimated are more likely to overstate the future annual compliance costs that industry will incur, rather than understate those costs.

# 7.13 Significant Energy Impact

According to E.O. 13211: Actions that Significantly Affect Energy Supply, Distribution, or Use, this rule is significant because it has a greater than 1 percent impact on the cost of natural gas and electricity production and it results in the retirement of greater than 500 MW of coal-fired generation.

Several aspects of CAIR are designed to minimize the impact on energy production. First, EPA recommends a trading program rather than the use of command-and-control regulations. Second, compliance deadlines are set cognizant of the impact that those deadlines have on electricity production. Both of these aspects of CAIR reduce the impact of the proposal on the electricity sector.

### 7.14 Industry-Sector Impacts

EPA estimates the direct costs of implementing CAIR at \$3.6 billion in 2015 in the CAIR region. Given the impact of this rule on electricity generators, we believe it is important to gauge the extent to which the rule might affect other industry sectors. To do so, we conducted a limited analysis of the economy-wide effects of implementing CAIR.

EPA was particularly interested in learning how anticipated changes in electricity prices might affect industry sectors that are large electricity users. The models we employed indicated those impacts would be small, even without incorporating the beneficial economic effects of CAIR-related air quality improvements such as improved worker health and productivity. Rather, our analyses continue to show that the value of even the limited subset of CAIR benefits we were able to quantify substantially outweigh implementation costs.

By focusing only on cost-side spillover effects on the economy, the industry-sector impacts projected by our macroeconomic models are likely overstated, primarily because the positive market impacts of CAIR on labor availability and productivity are excluded. In this regard, an independent panel of experts has encouraged EPA to work toward incorporating both EPA is working to develop this capability. Although neither model has yet been configured to include the indirect economic benefits

of air quality improvements, EPA employed two distinct computable general equilibrium models to gauge the potential magnitude of the economy-wide effects of CAIR implementation costs. The first model, known as IGEM, has a long track record and was used by the Agency for the first of the two Clean Air Act Section 812 studies. The other model, called EMPAX-CGE, is currently in peer review and has the advantage of disaggregating the United States into multiple regions. As with all models, these tools have their respective strengths and weaknesses, and differences in data and choice of functional form imply that the models are likely to show slightly different results. Despite the differences between the models, the results of the respective analyses show similarly small impacts of CAIR on energy-intensive industries. For example, production changes for the chemical manufacturing industry are estimated at -0.01 percent to -0.04 percent in 2010. Furthermore, if labor productivity improvements and other benefits of improved air quality were included, the small production output decreases projected by both models might be partially or entirely offset. Please see Appendix E for more details.

beneficial and costly effects when modeling the economy-wide consequences of regulation.

# 7.15 References

- Atkinson, S., and T. Tietenberg. 1982. "The Empirical Properties of Two Classes of Design for Transferable Discharge Permit Markets." *Journal of Environmental Economics and Management* 9:101-121
- Carlson, Curtis, Dallas R. Burtraw, Maureen, Cropper, and Karen L. Palmer. 2000. "Sulfur Dioxide Control by Electric Utilities: What Are the Gains from Trade?" *Journal of Political Economy* 108(#6):1292-1326.
- Ellerman, Denny. January 2003. Ex Post Evaluation of Tradable Permits: The U.S. SO₂ Cap-and-Trade Program. Massachusetts Institute of Technology Center for Energy and Environmental Policy Research.
- Harrington, W., R.D. Morgenstern, and P. Nelson. 2000. "On the Accuracy of Regulatory Cost Estimates." *Journal of Policy Analysis and Management* 19(2):297-322.
- Krupnick, A., W. Oates, and E. Van De Verg. 1980. "On Marketable Air Pollution Permits: The Case for a System of Pollution Offsets." *Journal of Environmental Economics and Management* 10:233-47.
- Manson, Nelson, and Neumann. 2002. "Assessing the Impact of Progress and Learning Curves on Clean Air Act Compliance Costs." Industrial Economics Incorporated.

McGartland, A., and W. Oates. 1985. "Marketable Permits for the Prevention of Environmental Deterioration." *Journal of Environmental Economics and Management* 12:207-228.

Montgomery, W. David. 1972. "Markets in Licenses and Efficient Pollution Control Programs." *Journal of Economic Theory* 5(3):395-418.

### **CHAPTER 8**

### STATUTORY AND EXECUTIVE ORDER IMPACT ANALYSES

This chapter presents discussion and analyses relating to relevant Executive Orders and statutory requirements relevant for CAIR. We discuss potential impacts to affected small entities as required by the Regulatory Flexibility Act (RFA), as amended by the Small Business Regulatory Enforcement Fairness Act (SBREFA). We also describe the analysis conducted to meet the requirements of the Unfunded Mandates Reform Act of 1995 (UMRA) that assess the impact of CAIR for state, local and Tribal governments and the private sector. Analyses conducted to comply with the Paperwork Reduction Act (PRA) are also discussed. In addition, we address the requirements of Executive Order (EO) 13045: Protection of Children from Environmental Health and Safety Risks; EO 13175: Consultation and Coordination with Indian Tribal Governments; and EO 12898: Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations. Discussion of Executive Order 13211: Actions that Significantly Affect Energy Supply, Distribution or Use is provided in Chapter 7 of this report.

### 8.1 Small Entity Impacts

The Regulatory Flexibility Act (5 U.S.C. § 601 et seq.), as amended by the Small Business Regulatory Enforcement Fairness Act (Public Law No. 104-121), provides that whenever an agency is required to publish a general notice of proposed rulemaking, it must prepare and make available an initial regulatory flexibility analysis, unless it certifies that the proposed rule, if promulgated, will not have "a significant economic impact on a substantial number of small entities" (5 U.S.C. § 605[b]). Small entities include small businesses, small organizations, and small governmental jurisdictions.

For the purposes of assessing the impacts of CAIR on small entities, a small entity is defined as:

 A small business according to the Small Business Administration size standards by the North American Industry Classification System (NAICS) category of the owning entity. The range of small business size standards for electric utilities is 4 billion kilowatt-hours of production or less;

- (2) a small government jurisdiction that is a government of a city, county, town, district, or special district with a population of less than 50,000; and
- (3) a small organization that is any not-for-profit enterprise that is independently owned and operated and is not dominant in its field.

Table 8-1 lists entities potentially affected by this proposed rule with applicable NAICS code. It is important to note that the proposed rule leaves states to decide which sources to control, such that states may choose to regulate source categories in addition to those listed in Table 8-1.

Category	NAICS Code ^b	Examples of Potentially Regulated Entities
Industry	221112	Fossil fuel-fired electric utility steam generating units.
Federal Government	221112°	Fossil fuel-fired electric utility steam generating units owned by the federal government.
State/Local/	221112°	Fossil fuel-fired electric utility steam generating units owned by municipalities.
Tribal Government	921150	Fossil fuel-fired electric utility steam generating units in Indian Country.

## Table 8-1. Potentially Regulated Categories and Entities^a

^a Include NAICS categories for source categories that own and operate electric generating units only.

^b North American Industry Classification System.

Federal, state, or local government-owned and operated establishments are classified according to the activity in which they are engaged.

Courts have interpreted the RFA to require a regulatory flexibility analysis only when small entities will be subject to the requirements of the rule.¹ This rule would not establish specific requirements applicable to small entities. Instead, it would require states to develop, adopt, and submit SIP revisions that would achieve the necessary  $SO_2$  and  $NO_x$  reductions, leaving to states the task of determining how and by which entities these reductions will be obtained. Because affected states would decide which sources to control and the extent of emissions reductions each selected source would have to achieve, EPA cannot definitively

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¹ See Michigan v. EPA, 213 F.3d 663, 668-69 (D.C. Cir. 2000), cert. den. 121 S.Ct. 225, 149 L.Ed.2d 135 (2001). An agency's certification need consider the rule's impact only on entities subject to the rule.

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project the effect of the proposed rule on small entities. Although not required, EPA conducts a general analysis of the potential impact of CAIR on small entities.

EPA examined the potential economic impacts to small entities associated with this rulemaking based on assumptions of how the affected states will implement control measures to meet their  $NO_x$  and  $SO_2$  budgets. This analysis assumes that all affected states choose to meet their budgets by controlling EGUs only. This analysis does not examine potential indirect economic impacts associated with CAIR, such as employment effects in industries providing fuel and pollution control equipment, or the potential effects of electricity price increases on industries and households.

As is noted in Chapter 7, EPA initially conducted IPM modeling for today's final action using a control strategy that is similar, but not identical to, the final CAIR requirements. The control strategy that EPA initially modeled included three additional States (Arkansas, Delaware and New Jersey) within the region and required these States to make annual SO₂ and NO_x reductions. While these three States are included in this analysis, they are not required to make annual reductions under the final CAIR (in the "Proposed Rules" section of today's Federal Register publication, EPA is publishing a proposal to include Delaware and New Jersey in the CAIR region for annual SO₂ and NO_x reductions). The implication of this is that total impacts on small entities are somewhat overstated in this analysis.

## 8.1.1 Identification of Small Entities

EPA used EGRID data as a basis for compiling the list of potentially affected small entities. EGRID is EPA's Emissions & Generation Resource Integrated Database, which contains emissions and resource mix data for virtually every power plant and company that generates electricity in the United States.² The data set contains detailed ownership and corporate affiliation information. For plants burning fossil fuel as the primary fuel, plant-level boiler and generator capacity, heat input, generation, and emissions data were aggregated by owner and then parent company. Entities with more than 4 billion kWh of annual electricity generation were removed from the list, as were municipal-owned entities serving a population greater than 50,000. Finally, for cooperatives, investor-owned utilities, and subdivisions that generate less than 4 billion kWh of electricity annually but may be part of a large entity, additional research on power sales, operating revenues, and other business activities was performed to make a final determination regarding size. Because the rule does

² eGRID is available at http://www.epa.gov/cleanenergy/egrid/download.htm.

not affect units with a generating capacity of 25 MW or less, small entities that do not own at least one generating unit with a capacity greater than 25 MW were dropped from the data set. According to EPA's analysis, approximately 185 small entities were exempted by this provision. Finally, small entities for which IPM does not project generation in 2010 or 2015 in the base case were omitted from the analysis because they are not projected to be operating and thus will not face the costs of compliance with CAIR. Four small municipal entities are omitted for this reason. After omitting entities for the reasons above, EPA identified a total of 75 potentially affected small entities, out of a possible 264. The number of potentially affected small entities by ownership type is listed in Table 8-2.

EGU	Number of Potentially	Total Net Compliance Cost (\$1999 millions)		Entitio Complia >1% of G	of Small es with nce Costs Generation enues	Number of Small Entities with Compliance Costs >3% of Generation Revenues		
Ownership Type	Affected Entities	2010	2015	2010	2015	2010	2015	
Cooperative	17	13.2	22.3	8	11	5	9	
Investor- Owned Utility	2	3.1	2.8	1	2	0	0	
Municipal	49	-28.2	- 9.1	15	29	15	19	
Subdivision	5	-15.2	-1.7	3	3	0	1	
Other	2	0.1	0.1	1	1	0	1	
Total	75	-27.0	14.4	28	46	20	30	

## Table 8-2. Projected Impact of CAIR on Small Entities

Note: The total number of potentially affected entities in this table excludes the 189 entities that have been dropped because they will not be affected by CAIR. Also, the total number of entities with costs greater than 1 percent or 3 percent of revenues includes only entities experiencing positive costs. A negative cost value implies that the group of entities experiences a net savings under CAIR.

Source: IPM and TRUM analysis

## 8.1.2 Overview of Analysis and Results

This section presents the methodology and results for estimating the impact on CAIR to small entities in 2010 and 2015 based on the following endpoints:

- annual economic impacts of CAIR on small entities and
- ratio of small entity impacts to revenues from electricity generation.

#### 8.1.2.1 Methodology for Estimating Impacts of CAIR on Small Entities

An entity can comply with CAIR through some combination of the following: installing retrofit technologies, purchasing allowances, switching to a cleaner fuel, or reducing emissions through a reduction in generation. Additionally, units with more allowances than needed can sell these allowances on the market. The chosen compliance strategy will be primarily a function of the unit's marginal control costs and its position relative to the marginal control costs of other units.

To attempt to account for each potential control strategy, EPA estimates compliance costs as follows:

$$C_{Compliance} = \Delta C_{Operating+Retrofit} + \Delta C_{Fuel} + \Delta C_{Allowances} + \Delta C_{Transaction} - \Delta R$$
(8.1)

where C represents a component of cost as labeled, and  $\Delta$  R represents the retail value of foregone electricity generation.

In reality, compliance choices and market conditions can combine such that an entity may actually experience a savings in any of the individual components of cost. Under CAIR, for example, EPA projects that the price of low-sulfur coal will fall as many units install scrubbers and switch away from low-sulfur coal to cheaper bituminous coal, such that many entities actually experience a reduction in fuel costs as a result of lower prices due to the demand shift. Similarly, although some units will forgo some level of electricity generation (and thus revenues) to comply, this impact will be lessened on these entities by the projected increase in electricity prices under CAIR as well as reductions in fuel costs, and those not reducing generation levels will see an increase in electricity revenues. Elsewhere, units burning high or medium sulfur coal might decide to pay relatively more for low-sulfur coal under CAIR and sell allowances on the market, in the hopes of negating some or all of their compliance cost. Because this analysis evaluates the total costs along each of the four compliance strategies laid out above for each entity, it inevitably captures savings or gains such as those described. As a result, what we describe as cost is really more of a measure of the net economic impact of the rule on small entities.

For this analysis, EPA used IPM-parsed output to estimate costs based on the parameters above, at the unit level. These impacts were then summed for each small entity, adjusting for ownership share. Net impact estimates were based on the following: operating and retrofit costs, sale or purchase of allowances, and the change in fuel costs or electricity generation revenues under CAIR relative to the base case. These individual components of compliance cost were estimated as follows:

- (1) **Operating and retrofit costs:** Using the IPM-parsed output for the base case and CAIR (http://www.epa.gov/airmarkets/epa-ipm/iaqr.html), EPA identified units that install control technology under CAIR and the technology installed. The equations for calculating retrofit costs were adopted from EPA's Technology Retrofit and Updating Model (TRUM). The model calculates the capital cost (in \$/MW); the fixed operation and maintenance (O&M) cost (in \$/MW-year); the variable O&M cost (in \$/MWh); and the total annualized retrofit cost for units projected to install FGD, SCR, or SNCR.
- (2) Sale or purchase of allowances: EPA estimated the value of initial SO₂ and NO_x allowance holdings. For SO₂, units were assumed to retain their Phase II allowance allocations as determined under EPA's 1998 reallocation of Acid Rain allowances, adjusted to reflect the 50 percent reduction in 2010 and 65 percent reduction in 2015 under CAIR. Because of the resources involved in compiling allowance-holding data, the value of banked SO₂ allowances was not considered in this analysis. The implication of this is that the annual net purchase of allowances may be overstated for some units. For NO_x, the state emission budgets were assumed to receive a share of the state NO_x emission budget equal to its share of the total state heat input for that year in the base case. This is a simplification of what is included in the model rule, which proposes allocating NO_x allowances based on heat input from 1999-2002.³ However, states can ultimately decide how to allocate NO_x allowances.

To estimate the value of allowances holdings, allocated allowances were subtracted from projected emissions, and the difference was then multiplied by the allowance prices projected by IPM for 2010 and 2015. Units were assumed to purchase or sell allowances to exactly cover their projected emissions under CAIR.

(3) Fuel costs: Fuel costs were estimated by multiplying fuel input (MMBtu) by region and fuel-type-adjusted fuel prices (\$/MMBtu) from TRUM. The change in fuel expenditures under CAIR was then estimated by taking the difference in fuel costs between CAIR and the base case.

³ A similar approach was used in regulatory impact analyses for the 126 FIP and NO_x SIP Call.

(4) Value of electricity generated: EPA estimated electricity generation by first estimating unit capacity factor and maximum fuel capacity. Unit capacity factor is estimated by dividing fuel input (MMBtu) by maximum fuel capacity (MMBtu). The maximum fuel capacity was estimated by multiplying capacity (MW) * 8,760 operating hours * heat rate (MMBtu/MWh). The value of electricity generated is then estimated by multiplying capacity (MW)*capacity factor*8,760*regional-adjusted retail electricity price (\$/MWh).

As discussed later in this analysis, the small entities projected to be affected by CAIR do not have to operate in a competitive market environment and thus should be able to pass compliance costs on to consumers. To somewhat account for this, we incorporated the projected regional-adjusted retail electricity price calculated under CAIR in our estimation of generation revenue under CAIR.

(5) Administrative costs: Because most affected units are already monitored as a result of other regulatory requirements, EPA considered the primary administrative cost to be transaction costs related to purchasing or selling allowances. EPA assumed that transaction costs were equal to 1.5 percent of the total absolute value of a unit's allowances. This assumption is based on market research by ICF Consulting.

# 8.1.2.2 Results

The potential impacts of CAIR on small entities are summarized in Table 8-2. All costs are presented in \$1999. EPA estimated the annualized net compliance cost to small entities to be approximately –\$27.0 million in 2010 and \$14.4 million in 2015.⁴ As discussed below, these negative net compliance costs in 2010 are largely due to higher electricity prices (and therefore higher generation revenue) and reduced low-sulfur coal prices under CAIR. Based on EPA analysis, small entities experiencing the greatest impact under CAIR in terms of cost as a share of revenue are those projected to both reduce output and purchase allowances.

⁴ Neither the costs nor the revenues of units that retire under CAIR are included in the impact estimates. Because these units are better off retiring under CAIR than continuing operation, the true cost of the rule on these units is not represented by our modeling. The true cost of CAIR for these units is the differential between their costs in the base case and the costs of meeting their customers' demand under the rule.

Furthermore, approximately 180 MW of small cooperative capacity (5 units of 61 in this analysis) are projected by IPM to be uneconomic to maintain under CAIR relative to the base case, as are approximately 265 MW of small municipality-owned capacity (6 units of 101 in this analysis). One plant owned by a cooperative is projected to be uneconomic, as is one plant owned by a municipality. Overall, about 445 MW of total small entity capacity, or 1.0 percent of total small entity capacity in the CAIR region, is projected to be uneconomic to maintain under CAIR relative to the base case. To put these numbers in context, of all affected capacity under CAIR, about 5.3 GW (1.7 percent) of coal-fired capacity is projected to be uneconomic to maintain relative to the base case. This comparison suggests that small entities should not be disproportionately affected by CAIR. In practice, units projected to be uneconomic to maintain may be "mothballed," retired, or kept in service to ensure transmission reliability in certain parts of the grid. Our IPM modeling is unable to distinguish between these potential outcomes. Notably, none of the units affected are likely to be in a competitive market environment and thus should be able to pass compliance costs on to consumers.

EPA further assessed the economic and financial impacts of the rule using the ratio of compliance costs to the value of revenues from electricity generation, focusing in particular on entities for which this measure is greater than 1 percent. Although this metric is commonly used in EPA impact analyses, it makes the most sense when as a general matter an analysis is looking at small businesses that operate in competitive environments. However, small businesses in the electric power industry often operate in a price-regulated environment where they are able to recover expenses through rate increases. Given this, EPA considers the 1 percent measure in this case a crude measure of the price increases these small entities will be asking of rate commissions or making at publicly owned companies.

Of the 75 small entities considered in this analysis, and 264 total small entities in the CAIR region, 28 entities may experience compliance costs greater than 1 percent of generation revenues in 2010, while 46 may in 2015. Entities that experience negative net costs under CAIR are excluded from these totals. These results do not fully account for the reality that none of these entities operate in a competitive market and thus should be able to recover all of their costs of complying with CAIR. It should also be emphasized that under CAIR, states, through their choice of NO_x allowance allocation methodologies, can potentially mitigate adverse affects of CAIR on small entities. The number of entities with compliance costs exceeding 3 percent of generation revenues is also included in Table 8-2.

The distribution across entities of economic impacts as a share of base case revenue is summarized in Table 8-3. Although the distributions of economic impacts on each ownership type are in general fairly tight, there are a few outliers for which the percentage of economic impacts as a share of revenue is either very low or very high relative to the capacity-weighted average. In the cases where entities are projected to experience negative net impacts that are a high percentage of revenues, these entities have units that are able to switch to a cheaper, lower-sulfur coal to comply with CAIR and are able to maintain or increase generation levels, thus increasing revenues. Additionally, entities in regions for which we project large electricity price increases relative to other regions tend to be among those at the lower end of the distribution. In the cases where entities are projected to experience positive net impacts that are a high percentage of revenues, these entities do not find it economic to retrofit and are unable to switch to a lower sulfur coal. Thus, these entities comply primarily by purchasing allowances and reducing generation.

EGU Ownership	Capacity-Weighted Average Economic Impacts as a % of Generation Revenues		М	lin	Max		
Туре	2010	2015	2010	2015	2010	2015	
Cooperative	1.0%	1.8%	-20.9%	-13.1%	11.5%	8.4%	
Investor-owned utility	1.6%	1.5%	0.4%	1.5%	2.0%	1.5%	
Municipal	-3.8%	-1.3%	-13.8%	-20.4%	17.2%	43.4%	
Subdivision	-0.1%	0.0%	- 80.0%	- 27.6%	1.9%	3.1%	
Other	-0.3%	-0.2%	-0.7%	-0.8%	1.9%	3.1%	
All	-0.2%	0.0%	-80.0%	-27.6%	17.2%	43.4%	

Table 8-3. Summary of Distribution of Economic Impacts of CAIR on Small Entit
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Source: IPM and TRUM analysis

The separate components of annualized costs to small entities under CAIR are summarized in Table 8-4. The most significant components of incremental cost to these entities under CAIR are retrofit and operating cost and allowance purchases. Fuel costs fall over all ownership groups, because of the combination of switching to bituminous coal, reduced overall fuel use, and lower prices for low-sulfur coal. Additionally, increases in electricity generation revenue are experienced over municipal, subdivision, and other, in 2010, and over all ownership types in 2015. This is due largely to the projected increase in electricity prices under CAIR.

EGU Ownership	Retrofit + Operating Cost		Net Purchase of Allowances		Fuel Cost		Lost Electricity Revenue		Administrative Cost	
Туре	2010	2015	2010	2015	2010	2015	2010	2015	2010	2015
Cooperative	4.1	15.7	35.2	54.0	-34.0	-34.8	7.5	-13.1	0.3	0.5
Investor-Owned Utility	-0.1	0.0	6.5	11.1	-6.0	-6.9	2.6	-1.5	0.0	0.1
Municipal	2.2	5.6	17.7	30.7	-18.7	-27.7	-29.6	-18.0	0.2	0.3
Subdivision	10.4	9.4	-1.6	1.7	-3.2	-8.4	-20.9	-4.5	0.05	0.06
Other	0.374	0.375	-0.146	-0.106	-0.138	-0.117	-0.007	-0.018	0.001	0.001

Table 8-4. Incremental Annualized Costs under CAIR Summarized by Ownership
Group and Cost Category (\$1999 millions)

Note: Numbers may not add to totals in Table 8-2 due to rounding.

Source: IPM and TRUM analysis.

### 8.1.3 Summary of Small Entity Impacts

EPA examined the potential economic impacts to small entities associated with this rulemaking based on assumptions of how the affected states will implement control measures to meet their emissions. While EPA concludes that the RFA as amended by SBREFA does not apply to CAIR, these impacts have been calculated to provide additional understanding of the nature of potential impacts, and additional information to the states as they revise SIPs to meet the emissions budgets set by this rulemaking.

Overall, about 445 MW of total small entity capacity, or 1.0 percent of total small entity capacity in the CAIR region, is projected to be uneconomic to maintain under CAIR relative to the base case. In practice, units projected to be uneconomic to maintain may be

"mothballed," retired, or kept in service to ensure transmission reliability in certain parts of the grid. Our IPM modeling is unable to distinguish between these potential outcomes.

Furthermore, of the 75 small entities potentially affected, and the 264 small entities in the CAIR region that are included in EPA's modeling, 28 may experience compliance costs in excess of 1 percent of revenues in 2010, and 46 may in 2015, based on our assumptions of how the affected states implement control measures to meet their emissions budgets as set forth in this rulemaking. Potentially affected small entities experiencing compliance costs in excess of 1 percent of revenues have some potential for significant impact resulting from implementation of CAIR. However, as noted above, it is EPA's position that because none of the affected entities currently operate in a competitive market environment, they should be able to pass the costs of complying with CAIR on to rate-payers. Furthermore, the decision to include only units greater than 25 MW in size exempts 185 small entities that would otherwise be potentially affected by CAIR.

Two other points should be considered when evaluating the impact of CAIR, specifically, and cap-and-trade programs more generally, on small entities. First, under CAIR, the cap-and-trade program is designed such that states determine how  $NO_x$  allowances are to be allocated across units. A state that wishes to mitigate the impact of the rule on small entities might choose to allocate  $NO_x$  allowances in a manner that is favorable to small entities.

# 8.2 Unfunded Mandates Reform Act (UMRA) Analysis

Title II of the UMRA of 1995 (Public Law 104-4)(UMRA) establishes requirements for federal agencies to assess the effects of their regulatory actions on state, local, and Tribal governments and the private sector. Under Section 202 of the UMRA, 2 U.S.C. 1532, EPA generally must prepare a written statement, including a cost-benefit analysis, for any proposed or final rule that "includes any Federal mandate that may result in the expenditure by State, local, and Tribal governments, in the aggregate, or by the private sector, of \$100,000,000 or more ... in any one year." A "Federal mandate" is defined under Section 421(6), 2 U.S.C. 658(6), to include a "Federal intergovernmental mandate" and a "Federal private sector mandate." A "Federal intergovernmental mandate," in turn, is defined to include a regulation that "would impose an enforceable duty upon State, Local, or Tribal governments," Section 421(5)(A)(i), 2 U.S.C. 658(5)(A)(i), except for, among other things, a duty that is "a condition of Federal assistance," Section 421(5)(A)(i)(I). A "Federal private sector mandate" includes a regulation that "would impose an enforceable duty upon the private sector," with certain exceptions, Section 421(7)(A), 2 U.S.C. 658(7)(A). Before promulgating an EPA rule for which a written statement is needed under Section 202 of the UMRA, Section 205, 2 U.S.C. 1535, of the UMRA generally requires EPA to identify and consider a reasonable number of regulatory alternatives and adopt the least costly, most cost-effective, or least burdensome alternative that achieves the objectives of the rule.

EPA prepared a written statement for the Supplemental Notice of Proposed Rulemaking (SNPR) consistent with the requirements of Section 202 of the UMRA. Furthermore, as EPA stated in the proposal, EPA is not directly establishing any regulatory requirements that may significantly or uniquely affect small governments, including Tribal governments. Thus, under CAIR, EPA is not obligated to develop under Section 203 of the UMRA a small government agency plan. Furthermore, in a manner consistent with the intergovernmental consultation provisions of Section 204 of the UMRA, EPA carried out consultations with the governmental entities affected by this rule.

For several reasons, EPA is not concluding that the requirements of the UMRA apply to CAIR. First, it is questionable whether a requirement to submit a SIP revision would constitute a federal mandate in any case. The obligation for a state to revise its SIP that arises out of Section 110(a) of the CAA is not legally enforceable by a court of law and at most is a condition for continued receipt of highway funds. Therefore, it is possible to view an action requiring such a submittal as not creating any enforceable duty within the meaning of Section 421(5)(9a)(I) of UMRA (2 U.S.C. 658 (a)(I)). Even if it did, the duty could be viewed as falling within the exception for a condition of federal assistance under Section 421(5)(a)(i)(I) of UMRA (2 U.S.C. 658(5)(a)(i)(I)).

As noted earlier, however, notwithstanding these issues, EPA prepared for the SNPR the statement that would be required by the UMRA if its statutory provisions applied, and EPA has consulted with governmental entities as would be required by the UMRA. While not required for CAIR, EPA analyzed the economic impacts of CAIR on government entities for informational purposes. This analysis does not examine potential indirect economic impacts associated with CAIR, such as employment effects in industries providing fuel and pollution control equipment, or the potential effects of electricity price increases on industries and households.

As is noted in Chapter 7, EPA initially conducted IPM modeling for today's final action using a control strategy that is similar, but not identical to, the final CAIR requirements. The control strategy that EPA initially modeled included three additional States (Arkansas, Delaware and New Jersey) within the region and required these States to

make annual  $SO_2$  and  $NO_x$  reductions. While these three States are included in this analysis, they are not required to make annual reductions under the final CAIR (in the "Proposed Rules" section of today's Federal Register publication, EPA is publishing a proposal to include Delaware and New Jersey in the CAIR region for annual  $SO_2$  and  $NO_x$  reductions). The implication of this is that total impacts on government-owned entities are somewhat overstated in this analysis.

# 8.2.1 Identification of Government-Owned Entities

Using eGRID data, EPA identified state- and municipality-owned utilities and subdivisions in the CAIR region. EPA then used IPM-parsed output to associate these plants with individual generating units. Entities that did not own at least one unit with a generating capacity of greater than 25 MW were omitted from the analysis because of their exemption from the rule. This exempts 179 entities owned by state or local governments. Additionally, government-owned entities for which IPM does not project generation in either 2010 or 2015 under the base case or CAIR were exempted from this analysis, because they are not projected to be operating and thus will not face the costs of compliance with CAIR. Five municipal entities were dropped from the analysis for this reason. Thus, EPA identified 81 state and municipality-owned utilities that are potentially affected by CAIR, out of a possible 265, which are summarized in Table 8-5.

EGU	Potentially	Projected Annualized Costs (\$1,000,000)		Governme with Co Costs	ber of ent Entities mpliance >1% of n Revenues	Number of Government Entities with Compliance Costs >3% of Generation Revenues	
Ownership Type	Affected Entities	2010	2015	2010	2015	2010	2015
Subdivision	5	-\$15.2	-\$1.7	3	3	0	1
State	7	-\$134.2	-\$110.5	0	2	0	0
Municipal	69	-\$162.5	-\$97.9	17	34	17	22
Total	81	-\$311.9	-\$210.2	20	39	17	23

Table 8-5. Summar	y of Potential Impacts on	<b>Government Entities under CAIR</b>

Note: The total number of potentially affected entities in this table excludes the 184 entities that have been dropped because they will not be affected by CAIR. Also, the total number of entities with costs greater than 1 percent or 3 percent of revenues includes only entities experiencing positive costs. A negative cost value implies that the group of entities experiences a net savings under CAIR.
 Source: IPM and TRUM analysis

### 8.2.2 Overview of Analysis and Results

After identifying potentially affected government entities, EPA estimated the impact of CAIR in 2010 and 2015 based on the following:

- total impacts of compliance on government entities and
- ratio of small entity impacts to revenues from electricity generation.

The financial burden to owners of EGUs under CAIR is composed of compliance and administrative costs. This section outlines the compliance and administrative costs for the 81 potentially affected government-owned units in the CAIR region.

## 8.2.2.1 Methodology for Estimating Impacts of CAIR on Government Entities

The primary burden on state and municipal governments that operate utilities under CAIR is the cost of installing control technology on units to meet  $SO_2$  and  $NO_x$  emission limits or the cost of purchasing allowances. However, an entity can comply with CAIR through any combination of the following: installing retrofit technologies, purchasing allowances, switching to a cleaner fuel, or reducing emissions through a reduction in generation. Additionally, units with more allowances than needed can sell these allowances on the market. The chosen compliance strategy will be primarily a function of the unit's marginal control costs and its position relative to the marginal control costs of other units.

To attempt to account for each potential control strategy, EPA estimates compliance costs as follows:

$$C_{Compliance} = \Delta C_{Operating+Retrofit} + \Delta C_{Fuel} + \Delta C_{Allowances} + \Delta C_{Transaction} - \Delta R$$
(8.2)

where C represents a component of cost as labeled, and  $\Delta R$  represents the retail value of foregone electricity generation.

In reality, compliance choices and market conditions can combine such that an entity may actually experience a savings in any of the individual components of cost. Under CAIR, for example, EPA projects that the price of low-sulfur coal will fall as many units install scrubbers and switch away from low-sulfur coal to cheaper bituminous coal, such that many entities burning low-sulfur coal actually experience a reduction in fuel costs as a result of the demand shift. Similarly, although some units will forgo some level of electricity generation (and thus revenues) to comply, this impact will be lessened on these entities by the projected increase in electricity prices under CAIR as well as reductions in fuel costs, while those not reducing generation levels will see an increase in electricity revenues. Elsewhere, units burning high- or medium-sulfur coal might decide to pay relatively more for low-sulfur coal under CAIR and sell allowances on the market, in the hopes of negating some or all of their compliance cost. Because this analysis evaluates the total costs along each of the four compliance strategies laid out above for each entity, it inevitably captures savings or gains such as those described. As a result, what we describe as cost is really more of a measure of the net economic impact of the rule on small entities.

In this analysis, EPA used IPM-parsed output for the base case and CAIR (http://www.epa.gov/airmarkets/epa-ipm/iaqr.html) to estimate compliance cost at the unit level. These costs were then summed for each small entity, adjusting for ownership share. Compliance cost estimates were based on the following: operating and retrofit costs, sale or purchase of allowances, and the change in fuel costs or electricity generation revenues under CAIR relative to the base case. These components of compliance cost were estimated as follows:

- (1) Retrofit and operating costs: Using the IPM-parsed output for the base case and CAIR, EPA identified units that install control technology under CAIR and the technology installed. The equations for calculating retrofit costs for SCR, SNCR, and FGD were adopted from EPA's TRUM. The model calculates the capital cost (in \$/MW), the fixed O&M cost (in \$/MW-year), the variable O&M cost (in \$/MWh), and the total annualized retrofit and operating cost by unit.
- (2) Sale or purchase of allowances: EPA estimated the value of initial SO₂ and NO_x allowance holdings. For SO₂, units were assumed to retain their Phase II allowance allocations as determined under EPA's 1998 reallocation of Acid Rain allowances, adjusted to reflect the 50 percent reduction in 2010 and 65 percent reduction in 2015 under CAIR. The value of banked SO₂ allowances was not considered in this analysis. Because the use of banked allowances is expected to be a significant compliance strategy, this analysis most likely overstates annualized compliance costs. For NO_x, the state emission budgets were assumed to receive a share of the state NO_x emission budget equal to its share of the total state heat input for that year in the base case. This is a simplification of what is included in the model rule, which proposes allocating NO_x allowances based on heat input from 1999 through

1992.⁵ However, states can ultimately decide how to allocate  $NO_x$  allowances.

To estimate the value of allowances holdings, allocated allowances were subtracted from projected emissions, and the difference was then multiplied by the allowance price projected by IPM. Units were assumed to purchase or sell allowances to exactly cover their projected emissions under CAIR.

- (3) Fuel costs: Fuel costs were estimated by multiplying fuel input (MMBtu) by region and fuel type-adjusted fuel prices (\$/MMBtu) from TRUM. The change in fuel expenditures under CAIR was then estimated by taking the difference in fuel costs between CAIR and the base case.
- (4) Value of electricity generated: EPA estimated electricity generation by first estimating the unit capacity factor and maximum fuel capacity. The unit capacity factor is estimated by dividing fuel input (MMBtu) by maximum fuel capacity (MMBtu). The maximum fuel capacity was estimated by multiplying capacity (MW) * 8,760 operating hours * heat rate (MMBtu/MWh). The value of electricity generated was then estimated by multiplying capacity (MW)*capacity factor*8,760*regional-adjusted retail electricity price (\$/MWh).
- (5) Administrative costs: Because most affected units are already monitored as a result of other regulatory requirements, EPA considered the primary administrative cost to be transaction costs related to purchasing or selling allowances. EPA assumed that transaction costs were equal to 1.5 percent of the total absolute value of a unit's allowances. This assumption is based on market research by ICF Consulting.

# 8.2.2.2 Results

A summary of economic impacts on government-owned entities is presented in Table 8-5. According to EPA's analysis, the total net economic impact on each category of government-owned entity (state- and municipality-owned utilities and subdivisions) is expected to be negative in both 2010 and 2015.⁶ IPM modeling of CAIR projects that approximately 340 MW (8 units of 219 in this analysis) of municipality-owned capacity

⁵A similar approach was used in impact analyses for the 126 FIP and NO_x SIP Call.

⁶All costs are reported in 1999 dollars.

would be uneconomic to maintain under CAIR, beyond what is projected in the base case. This represents about 0.4 percent of all subdivision, state, and municipality capacity in the CAIR region. For comparison, overall affected capacity under CAIR, about 5.3GW, or 1.7 percent of all coal-fired capacity is projected to be uneconomic to maintain relative to the base case. This comparison suggests that government entities should not face a disproportionate burden under CAIR. In practice, units projected to be uneconomic to maintain may be "mothballed," retired, or kept in service to ensure transmission reliability in certain parts of the grid. Our IPM modeling is unable to distinguish between these potential outcomes.

As was done for the small entities analysis, EPA further assessed the economic and financial impacts of the rule using the ratio of compliance costs to the value of revenues from electricity generation in the base case, also focusing specifically on entities for which this measure is greater than 1 percent.⁷ EPA projects that 20 government entities will have compliance costs greater than 1 percent of revenues from electricity generation in 2010, and 39 will in 2015. Entities that are projected to experience negative compliance costs under CAIR are not included in those totals. This approach is more indicative of a significant impact when an analysis is looking at entities operating in a competitive market environment. Government-owned entities do not operate in a competitive market environment and therefore will be able to recover expenses under CAIR through rate increases. Given this, EPA considers the 1 percent measure in this case a crude measure of the extent to which rate increases will be made at publicly owned companies.

The distribution across entities of economic impacts as a share of base case revenue is summarized in Table 8-6. For state-owned entities and subdivisions, the maximum economic impact as a share of base case revenues is approximately 3 percent. A few municipality-owned entities experience economic impacts that are significantly higher than the capacity-weighted average for this group. In the cases where entities are projected to experience positive net costs that are a high percentage of revenues, these entities do not find it economic to retrofit and are unable to switch to a lower-sulfur coal. Thus, these entities comply primarily through the purchase of allowances and reductions in generation.

⁷Neither the costs nor the revenues of units that retire under CAIR are included in this portion of the analysis. Because these units are better off retiring under CAIR than continuing operation, the true cost of the rule on these units is not represented by our modeling. The true cost of CAIR for these units is the differential between their costs in the base case and the costs of meeting their customers' demand under the rule.

	Capacity-Weighted Average Economic Impacts as a % of Generation Revenues		М	in	Max	
EGU Ownership Type	2010	2015	2010	2015	2010	2015
Sub-division	-3.6%	-2.0%	-80.0%	-27.6%	1.9%	3.1%
State	-5.2%	-3.9%	-11.4%	-10.2%	0.2%	2.8%
Municipal	-5.9%	-0.3%	-13.8%	-20.4%	17.2%	43.5%
All	-4.2%	-2.3%	-80.0%	-27.6%	17.2%	43.5%

 Table 8-6. Distribution of Economic Impacts on Government Entities under CAIR

Source: IPM and TRUM analysis

Additionally, a few entities are projected to experience negative net costs that are a high percentage of base case revenues. These entities have units that are able to switch to a cheaper, lower-sulfur coal to comply with CAIR and are able to maintain or increase generation levels, thus increasing revenues. Additionally, entities in regions for which we project large electricity price increases relative to other regions tend to be among those at the lower end of the distribution.

The various components of annualized incremental cost under CAIR to each group of government entities are summarized in Table 8-7. Overall, with the exceptions of subdivisions in 2010, each group is a net purchaser of allowances. Additionally, each group experiences both a reduction in fuel expenditures and an increase in electricity revenue under CAIR. Incremental fuel costs are negative because of the combination of a reduction in total coal use, switching to bituminous coal, and reduced low-sulfur coal prices under CAIR. Additionally, although total electricity generation by government entities falls slightly under CAIR, the total loss in revenues is more than exceeded by the revenue gains projected as a result of retail electricity prices rising under CAIR.

## 8.2.3 Summary of Government Entity Impacts

EPA examined the potential economic impacts on state and municipality-owned entities associated with this rulemaking based on assumptions of how the affected states will implement control measures to meet their emissions. Although EPA does not conclude that the requirements of the UMRA apply to CAIR, these impacts have been calculated to

EGU Ownership				Net Purchase of Allowances		Fuel Cost		Lost Electricity Revenue		Administrative Cost	
Туре	2010	2015	2010	2015	2010	2015	2010	2015	2010	2015	
Subdivision	10.4	9.4	-1.6	1.7	-3.2	-8.4	-20.9	-4.5	0.0	0.1	
State	20.1	25.9	29.2	52.5	-116.8	-143.1	-67.0	-46.3	0.3	0.5	
Municipal	21.3	26.7	39.3	94.8	-120.0	-156.8	-103.7	-63.7	0.7	1.0	

 Table 8-7. Incremental Annualized Costs under CAIR Summarized by Ownership

 Group and Cost Category (\$1,000,000)

Source: IPM and TRUM analysis

provide additional understanding of the nature of potential impacts and additional information to the states as they revise SIPs to meet the emissions budgets set by this rulemaking.

According to EPA's analysis, the total net economic impact on government-owned entities is expected to be negative in both 2010 and 2015. However, IPM modeling projects that about 340 MW of municipality-owned capacity (about 0.4 percent of all subdivision, state, and municipality capacity in the CAIR region) would be uneconomic to maintain under CAIR, beyond what is projected in the base case. In practice, units projected to be uneconomic to maintain may be "mothballed," retired, or kept in service to ensure transmission reliability in certain parts of the grid. Our IPM modeling is unable to distinguish between these potential outcomes.

Of the 81 government entities considered in this analysis and the 265 government entities in the CAIR region that are included in EPA's modeling, 20 may experience compliance costs in excess of 1 percent of revenues in 2010, and 39 may in 2015, based on our assumptions of how the affected states implement control measures to meet their emissions budgets as set forth in this rulemaking.

Government entities projected to experience compliance costs in excess of 1 percent of revenues have some potential for significant impact resulting from implementation of CAIR. However, as noted above, it is EPA's position that because these government entities can pass on their costs of compliance to rate-payers, they will not be significantly affected. Furthermore, the decision to include only units greater than 25 MW in size exempts 179 government entities that would otherwise be potentially affected by CAIR. The above points aside, potential adverse impacts of CAIR on state- and municipality-owned entities could be limited by the fact that the cap-and-trade program is designed such that states determine how  $NO_x$  allowances are to be allocated across units. A state that wishes to mitigate the impact of the rule on state- or municipality-owned entities might choose to allocate  $NO_x$  allowances in a manner that is favorable to these entities. Finally, in general, the use of cap-and-trade programs in general will limit impacts on entities owned by small governments relative to a less flexible command-and-control program.

## 8.3 Paperwork Reduction Act

In compliance with the Paperwork Reduction Act (44 U.S.C. 3501 <u>et seq</u>.), EPA submitted a proposed Information Collection Request (ICR) (EPA ICR number 2512.01) to the Office of Management and Budget (OMB) for review and approval on July 19, 2004 (FR 42720-42722). The ICR describes the nature of the information collection and its estimated burden and cost associated with the final rule. In cases where information is already collected by a related program, the ICR takes into account only the additional burden. This situation arises in states that are also subject to requirements of the Consolidated Emissions Reporting Rule (EPA ICR number 0916.10; OMB control number 2060-0088) or for sources that are subject to the Acid Rain Program (EPA ICR number 1633.13; OMB control number 2060-0258) or NO_x SIP Call (EPA ICR number 1857.03; OMB number 2060-0445) requirements.

EPA solicited comments on specific aspects of the information collection. The purpose of the ICR is to estimate the anticipated monitoring, reporting, and record-keeping burden estimates and associated costs for states, local governments, and sources that are expected to result from CAIR.

The record-keeping and reporting burden to sources resulting from states choosing to participate in a regional cap-and-trade program is approximately \$42 million annually. This estimate includes the annualized cost of installing and operating appropriate  $SO_2$  and  $NO_x$  emissions monitoring equipment to measure and report the total emissions of these pollutants from affected EGUs (serving generators greater than 25 megawatt electrical). The burden to state and local air agencies includes any necessary SIP revisions, performance of monitoring certification, and fulfilling of audit responsibilities. More information on the ICR analysis is included in the official CAIR docket.

In accordance with the Paperwork Reduction Act on July 19, 2004, an ICR was made available to the public for comment. The 60-day comment period expired September 19, 2004, with no public comments received specific to the ICR.

#### 8.4 Children's Health

E.O. 13045, "Protection of Children from Environmental Health Risks and Safety Risks" (62 FR 19885, April 23, 1997), applies to any rule that (1) is determined to be "economically significant" as defined under E.O. 12866 and (2) concerns an environmental health or safety risk that EPA has reason to believe may have a disproportionate effect on children. If the regulatory action meets both criteria, Section 5–501 of the Order directs the Agency to evaluate the environmental health or safety effects of the planned rule on children and explain why the planned regulation is preferable to other potentially effective and reasonably feasible alternatives considered by the Agency.

This final rule is not subject to this E.O., because it does not involve decisions on environmental health or safety risks that may disproportionately affect children. EPA believes that the emissions reductions from the strategies in this rule will further improve air quality and will further improve children's health. Chapter 4 of the RIA outlines benefits such as reduced incidences of respiratory illness, acute bronchitis, and asthma attacks for children that are anticipated to occur as a result of this rule.

#### 8.5 Tribal Impacts

E.O. 13175, entitled "Consultation and Coordination with Indian Tribal Governments" (65 FR 67249, November 9, 2000), requires EPA to develop an accountable process to ensure "meaningful and timely input by Tribal officials in the development of regulatory policies that have Tribal implications." This rule does not have "Tribal implications" as specified in E.O. 13175.

This rule addresses transport of pollutants that are precursor for ozone and  $PM_{2.5}$ . The CAA provides for states and Tribes to develop plans to regulate emissions of air pollutants within their jurisdictions. The regulations clarify the statutory obligations of states and Tribes that develop plans to implement this rule. The Tribal Authority Rule (TAR) gives Tribes the opportunity to develop and implement CAA programs, but it leaves to the discretion of the Tribe whether to develop these programs and which programs, or appropriate elements of a program, the Tribe will adopt.

This rule does not have Tribal implications as defined by E.O. 13175. It does not have a substantial direct effect on one or more Indian Tribes, because no Tribe has implemented an air quality management program at this time. Furthermore, this rule does not affect the relationship or distribution of power and responsibilities between the federal government and Indian Tribes. The CAA and the TAR establish the relationship of the federal government and Tribes in developing plans to attain the NAAQS, and this rule does nothing to modify that relationship. Because this rule does not have Tribal implications, E.O. 13175 does not apply.

If one assumes a Tribe is implementing a Tribal Implementation Plan, this final rule would have implications for that Tribe, but it would neither impose substantial direct costs on the Tribe nor preempt Tribal law. As provided above, EPA has estimated that the total annual private costs for the rule for the CAIR region as implemented by state, local, and Tribal governments is approximately \$2.4 billion in 2010 and \$3.6 billion in 2015 (1999\$). There are currently very few emissions sources in Indian country that could be affected by this rule and the percentage of Tribal land that will be impacted is very small. For Tribes that choose to regulate sources in Indian country, the costs would be attributed to inspecting regulated facilities and enforcing adopted regulations.

Although E.O. 13175 does not apply to this rule, EPA consulted with Tribal officials in developing this rule. EPA has encouraged Tribal input at an early stage. Also, EPA held periodic meetings with the states and the Tribes during the technical development of this rule. Three meetings were held with the Crow Tribe, where the Tribe expressed concerns about potential impacts of the rule on the coal mine operations. In addition, EPA held three calls with Tribal environmental professionals to address concerns specific to the Tribes. These discussions have given EPA valuable information about Tribal concerns regarding the development of this rule. EPA has provided briefings for Tribal representatives, and the newly formed National Tribal Air Association (NTAA), and other national Tribal forums. Input from Tribal representatives has been taken into consideration in developing this rule.

# 8.6 Environmental Justice

E.O. 12898, "Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations," requires federal agencies to consider the impact of programs, policies, and activities on minority populations and low-income populations. According to EPA guidance, agencies are to assess whether minority or low-income populations face risks or a rate of exposure to hazards that are significant and that "appreciably exceed or is likely to appreciably exceed the risk or rate to the general population or to the appropriate comparison group" (EPA, 1998).

In accordance with E.O. 12898, the Agency has considered whether this rule may have disproportionate negative impacts on minority or low income populations. The Agency expects this rule to lead to reductions in air pollution and exposures generally. For this reason, negative impacts to these subpopulations that appreciably exceed similar impacts to the general population are not expected.

# 8.7 Reference

U.S. Environmental Protection Agency (EPA). April 1998. *Guidance for Incorporating Environmental Justice Concerns in EPA's NEPA Compliance Analyses*. Washington, DC: Office of Federal Activities.

## **SECTION 9**

## **COMPARISON OF BENEFITS AND COSTS**

The estimated social costs to implement the final CAIR program, as described in this document, are approximately \$1.91 or \$2.14 billion annually for 2010 and \$2.56 or \$3.07 billion annually for 2015 (1999 dollars, 3 percent and 7 percent discount rate, respectively). Thus, the net benefits (social benefits minus social costs) of the program in 2010 are approximately \$71.4 + B billion or \$60.4 + B billion annually and in 2015 are \$98.5 + B billion or \$83.2 + B billion annually (1999 dollars, based on a discount rate of 3 percent and 7 percent, respectively). (B represents the sum of all unquantified benefits and disbenefits of the regulation.) Therefore, implementation of this rule is expected, based purely on economic efficiency criteria, to provide society with a significant net gain in social welfare, even given the limited set of health and environmental effects we were able to quantify. Addition of ozone-, directly emitted  $PM_{2.5}$ -, mercury-, acidification-, and eutrophication-related impacts would likely increase the net benefits of the rule. Table 9-1 presents a summary of the benefits, costs, and net benefits of the final rule.

The benefits and costs reported represent estimates for a complete CAIR program that includes the CAIR promulgated rule and the proposal to include annual  $SO_2$  and  $NO_x$  controls for New Jersey and Delaware. Annual  $SO_2$  and  $NO_x$  controls for Arkansas are included in the modeling used to develop these estimates resulting in a minimal overstatement of the reported benefits and costs for the complete CAIR program.

Air quality modeling was not conducted for the New Jersey and Delaware proposal. For this reason, an analysis of the potential benefits for the New Jersey and Delaware proposal could not be completed with any degree of specificity. However based on the air quality modeling results for the CAIR, we make rough estimates of the benefits and net benefits that might occur with this proposal. Including New Jersey and Delaware in the CAIR program would result in additional reductions of SO₂ and NO_x emissions. We estimate that approximately \$630 million of the total annual CAIR program benefits previously discussed may be attributable to annual SO₂ and NO_x controls for New Jersey and Delaware in 2010. This estimate increases to approximately \$1.1 billion annually in 2015. The full CAIR analysis including New Jersey and Delaware showed a benefit-cost ratio of

Description	2010	2015
Social costs ^b		
3 percent discount rate	\$1.91	\$2.56
7 percent discount rate	\$2.14	\$3.07
Social benefits ^{c,d,e}		
3 percent discount rate	73.3 + B	101 + B
7 percent discount rate	62.6 + B	86.3 + B
Health-related benefits:		
3 percent discount rate	72.1	99.3
7 percent discount rate	61.4	84.5
Visibility benefits	1.14	1.78
Net benefits (benefits-costs) ^{e,f}		
3 percent discount rate	\$71.4 + B	\$98.5 + B
7 percent discount rate	\$60.4 + B	\$83.2 + B

 Table 9-1.
 Summary of Annual Benefits, Costs, and Net Benefits of the Clean Air

 Interstate Rule^a (billions of 1999 dollars)

All estimates are rounded to three significant digits for ease of presentation and computation. Estimates represent a complete CAIR program that includes the CAIR promulgated rule and the proposal to include annual  $SO_2$  and  $NO_x$  controls for New Jersey and Delaware. Annual  $SO_2$  and  $NO_x$  controls for Arkansas are included in the modeling used to develop these estimates resulting in a minimal overstatement of the benefits and costs for the complete CAIR program.

^b Note that costs are the annualized total costs of reducing pollutants including NO_x and SO₂ for the EGU source category in the CAIR region in the years 2010 and 2015.

^c As this table indicates, total benefits are driven primarily by PM-related health benefits. The reduction in premature fatalities each year accounts for over 90 percent of total monetized benefits. Benefits in this table are nationwide (with the exception of ozone and visibility) and are associated with NO_x and SO₂ reductions. Ozone benefits represent benefits in the eastern United States. Visibility benefits represent benefits in Class I areas in the southeastern United States.

^d Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Potential benefit categories that have not been quantified and monetized are listed in Table 1-4.

^e Valuation assumes discounting over the SAB-recommended 20-year segmented lag structure described in Chapter 4. Results reflect the use of 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).

^f Net benefits are rounded to the nearest \$100 million. Columnar totals may not sum due to rounding.

around 39:1 in 2015. Based on the relatively low estimated private costs of including New Jersey and Delaware of \$30 million in 2010 and \$40 million in 2015, it is highly unlikely that costs of including New Jersey and Delaware would exceed benefits even if benefits of controlling  $SO_2$  and  $NO_x$  for New Jersey and Delaware were substantially lower than the average benefit we used to estimate the benefits. It is highly unlikely that benefits are much lower than average given the urban nature of much of New Jersey, and the proximity of New Jersey and Delaware to many heavily populated urban areas.

As with any complex analysis of this scope, there are several uncertainties inherent in the final estimate of benefits and costs, that are described fully in Chapters 4 and 7. In addition to the uncertainty characterization provided in these chapters, we also present two types of probabilistic approaches to characterize uncertainty in the benefit estimate of the CAIR program. The first approach generates a distribution of benefits based on the classical statistical error expressed in the underlying health and economic valuation studies used in the benefits modeling framework. The second approach uses the results from a pilot expert elicitation project designed to characterize key aspects of uncertainty in the ambient  $PM_{2.5}$ /mortality relationship, and augments the uncertainties in the mortality estimate with the statistical error reported for other endpoints in the benefit analysis.

## 9.1 References

- Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- U.S. Environmental Protection Agency (EPA). September 2000. *Guidelines for Preparing Economic Analyses*. EPA 240-R-00-003.
- U.S. Office of Management and Budget (OMB). 2003. Circular A-4 Guidance to Federal Agencies on Preparation of Regulatory Analysis.
- Woodruff, T.J., J. Grillo, and K.C. Schoendorf. 1997. "The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Infant Mortality and Particulate Air Pollution in the United States." *Environmental Health Perspectives* 105(6):608-612.

## **APPENDIX A**

# BENEFITS AND COSTS OF THE CLEAN AIR INTERSTATE RULE, THE CLEAN AIR VISIBILITY RULE, AND THE CLEAN AIR INTERSTATE RULE PLUS THE CLEAN AIR VISIBILITY RULE

This appendix presents the benefits and costs for the CAIR program (CAIR final rule plus the New Jersey and Delaware proposal),¹ the EGU requirements for Best Available Retrofit Technology (BART) Guidelines for the Regional Haze Rule, and the CAIR program in the CAIR region plus BART control for EGUs elsewhere in the country (CAIR Plus BART in the Non-CAIR Region). It is important to note that the CAIR, CAIR Plus BART in the Non-CAIR Region, and BART Nationwide benefit and costs estimates reflect controls for the EGU source category only, while the BART regulation will potentially affect 26 source categories. The analysis presented in this Appendix was conducted to show the benefits and costs of the alternative programs addressing the EGU sector.²

As Table A-1 shows, annual net benefits for the CAIR program are \$97.4 billion in 2015. This estimate compares to annual net benefits of \$44.3 billion for BART nationwide program and \$100 billion for CAIR Plus BART in the Non-CAIR Region (assuming a 3 percent discount rate). These estimates become \$82.7 billion for CAIR, \$84.9 billion for CAIR Plus BART in the Non-CAIR Region, and \$37.0 billion for the BART nationwide program assuming a 7 percent discount rate. The analysis shows that if one assumes the CAIR program exists, the incremental benefits of requiring BART controls for the EGU source category only in areas outside the CAIR region are approximately \$3.2 to \$4 billion (7 percent and 3 percent discount rate, respectively). Related incremental costs are approximately \$1 billion. All estimates are shown in 1999 dollars. Table A-2 lists the

¹ The modeling for the rule includes annual SO₂ and NO_x controls for Arkansas and results in a minimal overstatement of the benefits and costs of the CAIR program (CAIR final plus the New Jersey and Delaware proposal).

²Note that the net benefits reported in this appendix are estimated using the private costs of the respective rules rather than social costs. Thus the net benefits shown for the CAIR program in this appendix differ somewhat from the estimates presented in the body of this report.

Table A-1. Summary of Annual Benefits, Costs, and Net Benefits of the Clean Air Interstate Rule, 2015 (billions of 1999 dollars)^a

		CAIR Plus BART in the Non-CAIR	
Description	CAIR Program ^g	Region ^g	<b>BART Nationwide</b>
Private costs ^b	\$3.57	\$4.55	\$5.19
Social benefits ^{c,d,e}			
3 percent discount rate	\$101 + B	\$105 + B	\$49.5+B
7 percent discount rate	\$86.3 + B	\$89.5 + B	\$42.2 + B
Health-related benefits:			
3 percent discount rate	99.3	103	48.8
7 percent discount rate	84.5	87.6	41.5
Visibility benefits	1.78	1.89	0.699
Net benefits (benefits-costs) ^{e,f}			
3 percent discount rate	\$97.4 + B	100 + B	\$44.3 + B
7 percent discount rate	\$82.7 + B	\$84.9+ B	\$37.0 + B

^a All estimates are rounded to three significant digits for ease of presentation and computation. These annual estimates represent the benefits and costs of these regulatory programs expected to occur in 2015. BART estimates reflect benefits and costs for controls for the EGU source category only.

^b Note that costs presented are the annual total private costs to the power sector of reducing pollutants including  $NO_x$  and  $SO_2$ . The costs are estimated using the IPM and assume affected firms face cost of capital ranging from 5.34 to 6.74 percent. CAIR costs reflect costs for the CAIR region. Costs for CAIR Plus BART in the Non-CAIR Region and BART nationwide are national cost estimates.

^c Total benefits are driven primarily by PM-related health benefits. The reduction in premature fatalities each year accounts for over 90 percent of total monetized benefits in 2015. Benefits in this table are nationwide (with the exception of ozone and visibility) and are associated with  $NO_x$  and  $SO_2$  reductions. Ozone benefits relate to the eastern United States. Visibility benefits relate to Class I areas in the southeastern United States. While ozone benefits are expected for each of these programs, ozone benefits are included in the CAIR program benefits estimates only. The benefit estimates for CAIR Plus in the Non-CAIR Region BART and BART nationwide do not include ozone benefits. Inclusion of ozone benefits would not likely alter the conclusions reached on the magnitude of the difference between the options.

^d Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Potential effects categories that have not been quantified and monetized are listed in Table 1-4 and Table 1-5.

^e Valuation assumes discounting over the SAB-recommended 20-year segmented lag structure described in Chapter 4. Results reflect 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).

^f Net benefits are rounded to the nearest \$100 million. Columnar totals may not sum due to rounding.

^g CAIR costs and benefits are the estimates for the CAIR program that includes the promulgated CAIR and the proposal to include annual SO₂ and NO_x controls for New Jersey and Delaware. Modeling for CAIR assumes annual SO₂ and NO_x controls for Arkansas that is not a part of the CAIR program. Thus the benefits and costs reported are slightly overstated.

Health Effect	CAIR Program ^f	CAIR Plus BART in the Non-CAIR Region ^f	BART Nation- wide
PM-Related Endpoints:	*		
Premature mortality ^b Adult, age 30 and over Infant, age <1 year	17,000 36	17,000 38	8,200 19
Chronic bronchitis (adult, age 26 and over)	8,700	9,100	4,400
Nonfatal myocardial infarction (adults, age 18 and older)	22,000	23,000	11,000
Hospital admissions—respiratory (all ages) ^c	5,500	5,700	2,700
Hospital admissions—cardiovascular (adults, age >18) ^d	5,000	5,200	2,500
Emergency room visits for asthma (age 18 years and younger)	13,000	13,000	6,500
Acute bronchitis (children, aged 8-12)	19,000	20,000	10,000
Lower respiratory symptoms (children, aged 7-14)	230,000	240,000	120,000
Upper respiratory symptoms (asthmatic children, aged 9–18)	180,000	190,000	92,000
Asthma exacerbation (asthmatic children, aged 6-18)	290,000	310,000	150,000
Work loss days (adults, aged 18-65)	1,700,000	1,700,000	830,000
Minor restricted-activity days (adults, aged 18-65)	9,900,000	10,300,000	5,000,000
Ozone-Related Endpoints ^e			
Hospital admissions-respiratory causes (adult, 65 and older)	1,700	NE	NE
Hospital admissions—respiratory causes (children, under 2)	1,100	NE	NE
Emergency room visit for asthma (all ages)	280	NE	NE
Minor restricted-activity days (adults, aged 18-65)	690,000	NE	NE
School absence days	510,000	NE	NE

# Table A-2. Clean Air Interstate Rule: Estimated Reduction in Health Effects (Incidence)—2015

^a Incidences are rounded to two significant digits. BART estimates reflect incidences for controls for the EGU source category only.

^b Premature mortality benefits associated with ozone are not quantified in the primary analysis. Adult premature mortality estimates are based upon studies by Pope et al., 2002. Infant premature mortality estimates are based upon studies by Woodruff, Grillo, and Schoendorf, 1997.

^c Respiratory hospital admissions for PM include admissions for COPD, pneumonia, and asthma.

^d Cardiovascular hospital admissions for PM include total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

^e Although ozone benefits are expected to occur for CAIR Plus BART in the Non-CAIR Region and BART nationwide, ozone benefits are estimated for the CAIR program only.

^f These health effects incidences reflect estimates for the CAIR program (the promulgated CAIR and the proposal to include annual SO₂ and NO_x controls for New Jersey and Delaware in CAIR). Modeling for CAIR assumes annual SO₂ and NO_x controls for Arkansas that is not a part of the CAIR program. Thus the incidence estimates reported for CAIR are slightly overstated.

NE = Not estimated

reduction in health incidence resulting from the CAIR program, CAIR Plus BART in the Non-CAIR Region, and BART nationwide. Table A-3 depicts the monetary value of the benefit categories listed on Table A-2. We were unable to estimate all of the benefits and disbenefits associated with these rulemakings as summarized in Table 1-4. These unquantified effects are represented by the letter B.

Table A-3. Estimated Monetary Value in Reductions in Incidence of Health and
Welfare Effects (in millions of 1999\$)-2015 ^{a,b,c}

Health Effect	Pollutant	CAIR Program ^f	CAIR Plus BART in the Non-CAIR Region ^f	BART Nation- wide
Premature mortality ^d	1 onutant	1 Togram	Region	wide
Adult >30 years				
3% discount rate	PM _{2.5}	\$92,800	\$96,300	\$45,700
7% discount rate		78,100	81,100	38,400
Child <1 year		222	232	116
Chronic bronchitis (adults, 26 and over)	PM _{2.5}	3,340	3,510	1,690
Nonfatal acute myocardial infarctions				
3% discount rate	PM _{2.5}	1,850	1,920	905
7% discount rate		1,790	1,860	876
Hospital admissions for respiratory causes	$PM_{2.5}, O_3$	78.9	43.6	20.6
Hospital admissions for cardiovascular causes	PM _{2.5}	105	82.6	39.3
Emergency room visits for asthma	$PM_{2.5}, O_3$	3.56	3.62	1.79
Acute bronchitis (children, aged 8–12)	PM _{2.5}	7.06	7.41	3.68
Lower respiratory symptoms (children, 7–14)	PM _{2.5}	3.74	3.87	1.91
Upper respiratory symptoms (asthma, 9–11)	PM _{2.5}	4.77	4.69	2.30
Asthma exacerbations	PM _{2.5}	12.7	13.4	6.56
Work loss days	PM _{2.5}	219	209	101
Minor restricted-activity days (MRADs)	PM _{2.5} , O ₃	543	528	256
School absence days	$O_3$	36.4	NE	NE
Worker productivity (outdoor workers, 18–65)	$O_3$	19.9	NE	NE
Recreational visibility, 81 Class I areas	PM _{2.5}	1,780	1,890	699
Monetized Total ^e				
Base estimate:				
3% discount rate		\$101+B	\$105+B	\$49.5+
7% discount rate		\$86.3+B	\$89.5+B	В \$42.2+
				\$42.2+ B

Monetary benefits are rounded to three significant digits for ease of presentation and computation. Benefit estimates relate to emissions reductions for the EGU source category only. Estimates represent nationwide benefits (with the exception of ozone and visibility) and are associated with  $NO_x$  and  $SO_2$  reductions. Ozone benefits represent benefits for the eastern United States. Visibility estimates relate to Class I areas in the southeastern United States. BART estimates reflect benefits for controls for the EGU source category only. (continued)

# Table A-3. Estimated Monetary Value in Reductions in Incidence of Health and Welfare Effects (in millions of 1999\$)—2015^{a,b,c} (continued)

- ^b Monetary benefits adjusted to account for growth in real GDP per capita between 1990 and 2015.
- ^c Ozone benefits are estimated for the final CAIR. While ozone benefits are anticipated for CAIR plus BART in the Non-CAIR Region and BART nationwide, these ozone benefits were not estimated.
- ^d Valuation assumes discounting over the SAB-recommended 20-year segmented lag structure described earlier. Results show 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003). Adult premature mortality estimates are based upon studies by Pope et al., 2002. Infant premature mortality estimates are based upon studies by Woodruff, Grillo, and Schoendorf, 1997.
- ^e B represents the monetary value of health and welfare benefits not monetized. A detailed listing of unquantified benefits is provided in Table 1-4. Columnar totals may not add due to rounding.
- ^f These benefits reflect estimates for the CAIR program (the promulgated CAIR and the proposal to include annual  $SO_2$  and  $NO_x$  controls for New Jersey and Delaware in CAIR). Modeling for CAIR assumes annual  $SO_2$  and  $NO_x$  controls for Arkansas that is not a part of the CAIR program. Thus the benefit estimates reported for CAIR are slightly overstated.

NE = Not estimated

## A.1 References

- Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- U.S. Environmental Protection Agency (EPA). September 2000. *Guidelines for Preparing Economic Analyses*. EPA 240-R-00-003.
- U.S. Office of Management and Budget (OMB). 2003. Circular A-4 Guidance to Federal Agencies on Preparation of Regulatory Analysis.
- Woodruff, T.J., J. Grillo, and K.C. Schoendorf. 1997. "The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Infant Mortality and Particulate Air Pollution in the United States." *Environmental Health Perspectives* 105(6):608-612.

## **APPENDIX B**

# SUPPLEMENTAL ANALYSES ADDRESSING UNCERTAINTIES IN THE BENEFITS ANALYSES

#### **B.1** Introduction

The recent NAS report on estimating public health benefits of air pollution regulations recommended that EPA begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabilistic, multiple-source uncertainty analyses. In this appendix, we describe our progress toward improving our approach of characterizing the uncertainties in our economic benefits estimates, with particular emphasis on the C-R function relating premature mortality to exposures to ambient  $PM_{2.5}$ . We present two approaches to generating probabilistic distributions designed to illustrate the potential influence of some aspects of the uncertainty in the C-R function in a PM benefits analysis. The first approach generates a probabilistic estimate of statistical uncertainty based on standard errors reported in the underlying studies used in the benefit modeling framework. The second approach uses the results from a pilot expert elicitation designed to characterize certain aspects of uncertainty in the  $PM_{2.5}$ /mortality relationship.

In recent benefit analyses of air pollution regulations, estimation of the reduction in premature mortality from the control of particles accounts for 85 to 95 percent of total benefits. Therefore, it is an endpoint that will be an important focus for characterizing the uncertainty related to the estimates of total benefits. As part of a collaboration between EPA's Office of Air and Radiation (OAR) and the Office of Management and Budget (OMB) on the Nonroad Diesel Rule, we conducted a pilot expert elicitation intended to more fully characterize uncertainty in the estimate of mortality resulting from exposure to PM.

It should be recognized that in addition to uncertainty, the annual benefits estimates for the final CAIR also are inherently variable, due to the truly random processes that govern pollutant emissions and ambient air quality in a given year. Factors such as hourly rate of emissions and daily weather display constant variability regardless of our ability to accurately measure them.

## **B.1.1** General Approach

In addition to the two approaches to characterize uncertainty for PM mortality, we also wanted to incorporate information on uncertainties of other endpoints in the benefits model. For this rule we did not attempt to assign probabilities to all of the uncertain parameters in the model because of a lack of resources and reliable methods. At this time, we simply generate estimates of the distributions of dollar benefits for PM health effects and for total dollar benefits including visibility. For non-mortality endpoints, we provide a likelihood distribution for the total benefits estimate, based solely on the statistical uncertainty surrounding the estimated C-R functions and the assumed distributions around the unit values. Visibility benefits are also included in the estimate of total benefits, but because of data limitations, are characterized as a constant value rather than a distribution. This effectively shifts the distribution of total benefits upwards at all percentiles by the same amount.

Our estimate of the likelihood distribution for total benefits should be viewed as incomplete because of the wide range of sources of uncertainty that we have not incorporated. The 5th and 95th percentile points of our estimate are based on statistical error, and cross-study variability provides some insight into how uncertain our estimate is with regard to those sources of uncertainty. However, it does not capture other sources of uncertainty regarding other inputs to the model, including emissions, air quality, baseline population incidence, projected exposures, or the model itself, including aspects of the health science not captured in the studies, such as the likelihood that PM is causally related to premature mortality and other serious health effects and the likelihood that ozone has an independent effect on mortality. Thus, a likelihood description based on the standard error would provide a misleading picture about the overall uncertainty in the estimates.

Both the uncertainty about the incidence changes¹ and uncertainty about unit dollar values can be characterized by *distributions*. Each "likelihood distribution" characterizes our beliefs about what the true value of an unknown variable (e.g., the true change in incidence of a given health effect in relation to PM exposure) is likely to be, based on the available

¹ Because this is a national analysis in which, for each endpoint, a single C-R function is applied everywhere, there are two sources of uncertainty about incidence: statistical uncertainty (due to sampling error) about the true value of the pollutant coefficient in the location where the C-R function was estimated and uncertainty about how well any given pollutant coefficient approximates  $\beta^*$ .

information from relevant studies.² Unlike a sampling distribution (which describes the possible values that an *estimator* of an unknown variable might take on), this likelihood distribution describes our beliefs about what values the unknown variable itself might be. Such likelihood distributions can be constructed for each underlying unknown variable (such as a particular pollutant coefficient for a particular location) or for a function of several underlying unknown variables (such as the total dollar benefit of a regulation). In either case, a likelihood distribution is a characterization of our beliefs about what the unknown variable (or the function of unknown variables) is likely to be, based on all the available relevant information. A likelihood description based on such distributions is typically expressed as the interval from the 5th percentile point of the likelihood distribution to the 95th percentile point. If all uncertainty had been included, this range would be the "credible range" within which we believe the true value is likely to lie with 90 percent probability.

# **B.2** Monte-Carlo Based Uncertainty Analysis

The uncertainty about the total dollar benefit associated with any single endpoint combines the uncertainties from these two sources (the C-R relationship and the valuation) and is estimated with a Monte Carlo method. In each iteration of the Monte Carlo procedure, a value is randomly drawn from the incidence distribution, another value is randomly drawn from the incidence distribution, another value is randomly drawn from the total dollar benefit for that iteration is the product of the two.³ When this is repeated for many (e.g., thousands of) iterations, the distribution of total dollar benefits associated with the endpoint is generated.

Using this Monte Carlo procedure, a distribution of dollar benefits can be generated for each endpoint. As the number of Monte Carlo draws gets larger and larger, the Monte Carlo-generated distribution becomes a better and better approximation of a joint likelihood distribution (for the considered parameters) making up the total monetary benefits for the endpoint.

After endpoint-specific distributions are generated, the same Monte Carlo procedure can then be used to combine the dollar benefits from different (nonoverlapping) endpoints to generate a distribution of total dollar benefits.

² Although such a "likelihood distribution" is not formally a Bayesian posterior distribution, it is very similar in concept and function (see, for example, the discussion of the Bayesian approach in Kennedy (1990), pp. 168-172).

³ This method assumes that the incidence change and the unit dollar value for an endpoint are stochastically independent.

The estimate of total benefits may be thought of as the end result of a sequential process in which, at each step, the estimate of benefits from an additional source is added. Each time an estimate of dollar benefits from a new source (e.g., a new health endpoint) is added to the previous estimate of total dollar benefits, the estimated total dollar benefits increases. However, our bounding or likelihood description of where the true total value lies also increases as we add more sources.

As an example, consider the benefits from reductions in PM-related hospital admissions for cardiovascular disease. Because the actual dollar value is unknown, it may be described using a variable, with a distribution describing the possible values it might have. If this variable is denoted as  $X_1$ , then the mean of the distribution,  $E(X_1)$  and the variance of  $X_1$ , denoted  $Var(X_1)$ , and the 5th and 95th percentile points of the distribution (related to  $Var(X_1)$ ), are ways to describe the likelihood for the true but unknown value for the benefits reduction.

Now suppose the benefits from reductions in PM-related hospital admissions for respiratory diseases are added. Like the benefits from reductions in PM-related hospital admissions for cardiovascular disease, the likelihood distribution for where we expect the true value to be may be considered a variable, with a distribution. Denoting this variable as  $X_2$ , the benefits from reductions in the incidence of *both* types of hospital admissions is  $X_1 + X_2$ . This variable has a distribution with mean  $E(X_1 + X_2) = E(X_1) + E(X_2)$ , and a variance of Var $(X_1 + X_2) = Var(X_1) + Var(X_2) + 2Cov(X_1,X_2)$ ; if  $X_1$  and  $X_2$  are stochastically independent, then it has a variance of  $Var(X_1 + X_2) = Var(X_1) + Var(X_2)$ , and the covariance term is zero.

The benefits from reductions in all nonoverlapping PM-related health and welfare endpoints are  $(X_{m+1}, ..., X_n)$  is  $X = X_1 + ... + X_n$ . The mean of the distribution of total benefits, X, is

$$E(X) = E(X_1) + E(X_2) + \dots + E(X_n)$$
(B.1)

and the variance of the distribution of total benefits—assuming that the components are stochastically independent of each other (i.e., no covariance between variables)—is

$$Var(X) = Var(X_1) + Var(X_2) + ... + Var(X_n)$$
 (B.2)

If all the means are positive, then each additional source of benefits increases the point estimate (mean) of total benefits. However, with the addition of each new source of benefits, the variance of the estimate of total benefits also increases. That is,

$$E(X_1) < E(X_1 + X_2) < E(X_1 + X_2 + X_3) < \dots < E(X_1 + \dots + X_n) = E(X)$$
(B.3)

$$Var(X_1) < Var(X_1 + X_2) < Var(X_1 + X_2 + X_3) < \dots < Var(X_1 + \dots + X_n) = Var(X).$$
 (B.4)

That is, the addition of each new source of benefits results in a larger mean estimate of total benefits (as more and more sources of benefits are included in the total) about which there is less certainty. This phenomenon occurs whenever estimates of benefits are added.

Calculated with a Monte Carlo procedure, the distribution of X is composed of random draws from the components of X. In the first draw, a value is drawn from each of the distributions,  $X_1$ ,  $X_2$ , through  $X_n$ ; these values are summed; and the procedure is repeated again, with the number of repetitions set at a high enough value (e.g., 5,000) to reasonably trace out the distribution of X. The 5th percentile point of the distribution of X will be composed of points pulled from all points along the distributions of the individual components and not simply from the 5th percentile. Although the sum of the 5th percentiles of the components would be represented in the distribution of X generated by the Monte Carlo, it is likely that this value would occur at a significantly lower percentile. For a similar reason, the 95th percentile of X will be *less* than the sum of the 95th percentiles of the component values that are significantly lower than the 95th percentiles.

The physical effects estimated in this analysis are assumed to occur independently. It is possible that, for any given pollution level, there is some correlation between the occurrence of physical effects, due to say avoidance behavior or common causal pathways and treatments (e.g., stroke, some kidney disease, and heart attack are related to treatable blood pressure). Estimating accurately any such correlation, however, is beyond the scope of this analysis, and instead it is simply assumed that the physical effects occur independently.

We conducted two different Monte Carlo analyses, one based on the distribution of reductions in premature mortality characterized by the mean effect estimate and standard error from the epidemiology study of PM-associated mortality associated with the primary estimate in Chapter 4 (Pope et al., 2002), and one based on the results from a pilot expert elicitation project (IEc, 2004). In both analyses, the distributions of all other health endpoints are characterized by the reported mean and standard deviations from the epidemiology literature. Distributions for unit dollar values are based on reported ranges or distributions of values in the economics literature and are summarized in Table B-1. We are unable at this time to characterize the uncertainty in the estimate of benefits of improvements in visibility at Class I areas. As such, we treat the visibility benefits as fixed and add them to

	Mean Value,	
	Adjusted for	
	Income Growth to	
Health Endpoint	2030	Derivation of Distribution
Premature Mortality (Value of a Statistical Life)	\$5,500,000	Normal distribution with mean of \$5.5 million and standard deviation of \$2.3 million, anchored at 2.5 th and 97.5 th percentiles of \$1 and \$10 million, respectively. Confidence interval is based on two meta-analyses of the wage-risk VSL literature. \$1 million represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis. \$10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. The VSL represents the value of a small change in mortality risk aggregated over the affected population. Normal distribution chosen through best professional judgment.
Chronic Bronchitis (CB)	\$430,000	The WTP to avoid a case of pollution-related CB is calculated as $WTP_x = WTP_{13} * e^{-\beta^*(13-x)}$ , where x is the severity of an average CB case, WTP ₁₃ is the WTP for a severe case of CB, and $\beta$ is the parameter relating WTP to severity, based on the regression results reported in Krupnick and Cropper (1992). The distribution of WTP for an average severity-level case of CB was generated by Monte Carlo methods, drawing from each of three distributions: (1) WTP to avoid a severe case of CB is assigned a 1/9 probability of being each of the first nine deciles of the distribution of WTP responses in Viscusi et al. (1991); (2) the severity of a pollution- related case of CB (relative to the case described in the Viscusi study) is assumed to have a triangular distribution, with the most likely value at severity level 6.5 and endpoints at 1.0 and 12.0; and (3) the constant in the elasticity of WTP with respect to severity is normally distributed with mean = 0.18 and standard deviation = 0.0669 (from Krupnick and Cropper [1992]). This process and the rationale for choosing it is described in detail in the <i>Costs and</i> <i>Benefits of the Clean Air Act, 1990 to 2010</i> (EPA, 1999).

# Table B-1. Distributions for Unit Values of Health Endpoints

(continued)

#### Table B-1. Distributions for Unit Values of Health Endpoints (continued)

Health Endpoint	Mean Value, Adjusted for Income Growth to 2030	Derivation of Distribution		
Nonfatal Myocardial Infarction (heart attack) <u>3% discount rate</u> Age 0–24 Age 25–44	\$66,902	No distribution available. Age-specific COI values reflecting lost earnings and direct medical costs over a 5-year period following a nonfatal MI. Lost earnings estimates based on Cropper and Krupnick (1990). Direct medical costs based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990).		
Age 45–54 Age 55–65 Age 66 and over	\$74,676 \$78,834 \$140,649 \$66,902	Lost earnings: Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings: age of onset at 3%at 7%		
7% discount rate Age 0–24 Age 25–44 Age 45–54 Age 55–65 Age 66 and over	\$65,293 \$73,149 \$76,871 \$132,214 \$65,293	25-44       \$8,774       \$7,855         45-54       \$12,932       \$11,578         55-65       \$74,746       \$66,920         Direct medical expenses:       An average of:         1.       Wittels et al. (1990) (\$102,658—no discounting)         2.       Russell et al. (1998), 5-year period. (\$22,331 at 3% discount rate; \$21,113 at 7% discount rate)		
Hospital Admissions All Respiratory (ICD codes 480-487, 490-492, 494-496)	\$12,378	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).		
All Cardiovascular (ICD codes 390-429)	\$18,387	No distribution available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).		

(continued)

Table B-1.	Distributions	for Unit	Values of	Health	Endpoints	(continued)
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	Mean Value, Adjusted for	
	Income Growth to	
Health Endpoint	2030	Derivation of Distribution
Emergency Room Visits for Asthma	\$286	No distribution available. The COI point estimate is the simple average of two unit COI values: (1) \$311.55, from Smith et al. (1997) and
		(2) \$260.67, from Stanford et al. (1999).
Respiratory Ailments No	t Requiring Hospitalizat	tion
Upper Respiratory Symptoms (URS)	\$27	Combinations of the three symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in seven different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, we assumed a uniform distribution between \$10 and \$45.
Lower Respiratory Symptoms (LRS)	\$17	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS. In the absence of information surrounding the frequency with which each of the 11 types of LRS occurs within the LRS symptom complex, we assumed a uniform distribution between \$8 and \$25.
Asthma Exacerbations	\$45	Asthma exacerbations are valued at \$45 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma exacerbation is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. The value is assumed have a uniform distribution between \$17 and \$73.

(continued)

#### Table B-1. Distributions for Unit Values of Health Endpoints (continued)

Health Endpoint	Mean Value, Adjusted for Income Growth to 2030	Derivation of Distribution
Acute Bronchitis	\$390	Assumes a 6-day episode, with the distribution of the daily value specified as uniform with the low and high values based on those recommended for related respiratory symptoms in Neumann et al. (1994). The low daily estimate of \$10 is the sum of the mid-range values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high daily estimate was taken to be twice the value of a minor respiratory restricted-activity day, or \$110.
<b>Restricted Activity, Work</b>	x Loss, and School Abse	nce Days
School Absences	\$75	No distribution available. Point estimate is based on (1) the probability that if a school child stays home from school, a working mother will have to stay home from work to care for the child (0.73); and (2) lost productivity at the female parent's wage (average of \$103).
Work Loss Days (WLDs)	Variable	No distribution available. Point estimate is based on county- specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5—to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.
Minor Restricted- Activity Days (MRADs)	\$55	Median WTP estimate to avoid one MRAD from Tolley et al. (1986). Distribution is assumed to be triangular with a minimum of \$22 and a maximum of \$83, with a most likely value of \$55. Range is based on assumption that value should exceed WTP for a single mild symptom (the highest estimate for a single symptom—for eye irritation—is \$16.00) and be less than that for a WLD. The triangular distribution acknowledges that the actual value is likely to be closer to the point estimate than either extreme.

all percentiles of the PM health benefits distribution. Results of the Monte Carlo analysis based on the Pope et al. (2002) distribution are presented in the next section. Results of the Monte Carlo analysis based on the pilot expert elicitation are presented in Section B.3.

#### **B.2.1** Monte Carlo Analysis Using Classical Statistical Sources of Uncertainty

Based on the Monte Carlo techniques described earlier, we generated likelihood distributions for the dollar value of total annual benefits including PM health, ozone health, and visibility benefits for the final CAIR. For this analysis, the likelihood descriptions for the true value for each of the health endpoint incidence measures, including premature

mortality, were based on classical statistical uncertainty measures, including the mean and standard deviation for the C-R relationships in the epidemiological literature, and assumption of particular likelihood distribution shapes for the valuation for each health endpoint value based on reported values in the economic literature. Table B-1 summarizes the chosen parameters for likelihood distributions for unit values for each of the PM health effects included in the Monte Carlo simulation. The distributions for the value used to represent incidence of a health effect in the total benefits valuation represent both the simple statistical uncertainty surrounding individual effect estimates and, for those health endpoints with multiple effects from different epidemiology studies, interstudy variability. Visibility benefits are also included in the distribution of total benefits; however, we were unable to characterize a distribution for visibility benefits. As such, they are simply added to each percentile of the distribution of health benefits.

Results of the Monte Carlo simulations are presented in Table B-2. The table provides the estimated means of the distributions and the estimated 5th and 95th percentiles of the distributions. The contribution of mortality to the mean benefits and to both the 5th and 95th percentiles of total benefits is substantial, with mortality accounting for 93 percent of the mean estimate, and even the 5th percentile of mortality benefits dominating the 95th percentile of all other benefit categories. Thus, the choice of value and the shape for likelihood distribution for VSL should be examined closely and is key information to provide to decision makers for any decision involving this variable. The 95th percentile of total benefits is approximately twice the mean, while the 5th percentile is approximately one-fourth of the mean. The overall range from 5th to 95th represents about one order of magnitude.

#### **B.3** Pilot Expert Elicitation of PM Mortality Uncertainty

Expert elicitation is a formal, highly structured and well-documented process whereby expert judgments, usually of multiple experts, are obtained. Formal expert elicitation usually involves experts with training and expertise in statistics, decision analysis, and probability encoding who work with subject matter experts to structure questions about uncertain relationships or parameters and who design and implement the process used to obtain probability and other judgments from subject matter experts. Several academic traditions—judgment and decision-making, human factors, cognitive sciences, expert systems, management science, to name a few—have sought to understand how to successfully elicit probabilistic judgments from both lay people and experts (Morgan and Henrion, 1990; Cooke, 1991; Wright and Ayton, 1994; Ayyub, 2002). Over the past 2 decades, an increasing number of studies have used expert judgment techniques to characterize uncertainty in quantities of interest to environmental risk analysis and decision

### Table B-2. Distribution of Value of Annual Human Health and Welfare Benefits in 2015 for the Final CAIR Rule^a

	Monetary Benefits ^{b, c} (Millions 2000\$, Adjusted for Income Growth)			(Crowth)
	5 th		95 th	
Endpoint	Pollutant	Percentile	Mean	Percentile
Premature mortality ^c				
Long-term exposure, (adults, >30yrs)	PM	\$22,000	\$93,000	\$190,000
Long-term exposure (child <1yr)	PM	\$56	\$220	\$440
Chronic bronchitis (adults, 26 and over)	PM	\$160	\$3,300	\$11,000
Nonfatal myocardial infarctions	PM	\$440	\$1,800	\$4,100
Hospital admissions from respiratory causes	O ₃ and PM	\$30	\$79	\$140
Hospital admissions from cardiovascular causes	PM	\$64	\$110	\$150
Emergency room visits for asthma	O ₃ and PM	\$2.0	\$3.6	\$5.3
Acute bronchitis (children, aged 8-12)	PM	(\$0.2)	\$7.1	\$17
Lower respiratory symptoms (children, aged 7-14)	PM	\$1.4	\$3.7	\$6.9
Upper respiratory symptoms (asthmatic children, aged 9–11)	PM	\$1.1	\$4.8	\$10
Asthma exacerbations	PM	\$0.1	\$13	\$34
Work loss days (adults, aged 18–65)	PM	\$190	\$220	\$250
Minor restricted-activity days (adults, aged 18-65)	O ₃ and PM	\$320	\$550	\$790
School absence days (children, aged 6-11)	$O_3$	\$12	\$36	\$75
Worker productivity (outdoor workers, aged 18–65)	O ₃	\$20	\$20	\$20
Recreational visibility (81 Class I areas)	PM	\$1,800	\$1,800	\$1,800
Monetized Total ^d	O ₃ and PM	\$26,000+B	\$100,000+B	\$210,000+B

^a Monetary benefits are rounded to two significant digits.

^b Monetary benefits are adjusted to account for growth in real GDP per capita between 1990 and 2015.

^c Results show 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).

^d B represents the monetary value of the nonmonetized health and welfare benefits. A detailed listing of unquantified PM-, ozone-, and mercury-related health effects is provided in Table 4-2.

making. North and Merkhofer (1976) considered using expert judgment in evaluating emission control strategies. As referred to by the NAS, EPA's Office of Air Quality Planning and Standards (OAQPS) successfully used expert judgment to characterize uncertainty in the health effects of exposure to lead (McCurdy and Richmond, 1983; Whitfield and Wallsten, 1989) and to ozone (Whitfield et al., 1991; Winkler et al., 1995). Amaral (1983) and Morgan et al. (1984) used expert judgment to evaluate the transport and impacts of sulfur air pollution. Several studies have been done in the area of climate change (Manne and Richels, 1994; Nordhaus, 1994; Morgan and Keith, 1995; Reilly et al., 2001). Hawkins and Evans (1989) used industrial hygienists to predict toluene exposures to workers involved in a batch chemical process. In a more recent use of expert judgment in exposure analysis, Walker et al. (2001, 2003) asked experts to estimate ambient, indoor and personal air concentrations of benzene. A few studies have used expert judgment to characterize uncertainty in chemical dose-response: Hawkins and Graham (1988) and Evans et al. (1994a) for formaldehyde and Evans et al. (1994b) for risk of exposure to chloroform in drinking water. Expert judgment has also been used to characterize residential radon risks (Krewski et al., 1999). The Non-Road Diesel Rule (Mansfield, 2004) was the first illustration of an application of the results of an expert elicitation study to a regulatory policy analysis.

In its 2002 report, the NAS provides a number of recommendations for how EPA might improve the characterization of uncertainty in its benefits analyses. One recommendation was that:

"EPA should begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabilistic, multiple-source uncertainty analyses. This shift will require specification of probability distributions for major sources of uncertainty. These distributions should be based on available data and expert judgment" (NAS, 2002, p. 14). The NAS elaborated on this recommendation by saying "although the specific methods for selection and elicitation of experts may need to be modified somewhat, the protocols that have been developed and tested by OAQPS [in prior EPA projects—see below] provide a solid foundation for future work in the area. EPA may also consider having its approaches reviewed by decision analysts, biostatisticians, and psychologists from other fields where expert judgment is applied" (NAS, 2002, p. 140).

They recommended the use of formally elicited expert judgments but noted that a number of issues must be addressed and that sensitivity analyses would be needed for distributions that are based on expert judgment. They also recommended that EPA clearly distinguish between

data-derived components of an uncertainty assessment and those based on expert opinions. As a first step in addressing the NAS recommendations regarding expert elicitation, EPA, in collaboration with OMB, conducted a pilot expert elicitation to characterize uncertainties in the relationship between ambient  $PM_{2.5}$  and mortality.

#### **B.3.1** Elicitation Method

This pilot was designed to provide EPA with an opportunity to improve its understanding of the design and application of expert elicitation methods to economic benefits analysis and lay the groundwork for a more comprehensive elicitation. The scope of the pilot was limited to a one-year period to allow for inclusion in the Non-Road Diesel Rule. As such, we focused the elicitation on the C-R function of PM mass rather than on individual issues surrounding an estimate of the change in mortality due to PM exposure. We selected experts for participation from two previously established expert panels of the NAS. Due to time constraints, we chose not to conduct a workshop with the experts prior to the elicitation, which is often included in the protocol of elicitations to help condition and prepare the experts for the elicitation. A full description of the pilot is contained in a report titled "An Expert Judgment Assessment of the Concentration-Response Relationship between PM_{2.5} Exposure and Mortality" (IEc, 2004) available in the public docket of the Non-Road Diesel Rule.

The analytic plan for the pilot was developed based on established elicitation methods as suggested by the NAS and published in the peer-reviewed literature. The plan and protocol were reviewed in three separate contexts. It was internally reviewed by EPA and OMB scientists with experience using expert elicitation methods. The project team that implemented the pilot consisted of individuals with experience in expert elicitation and individuals with expertise in PM health effects and health benefits. Second, as part of a review of the analytical blueprint of EPA's Second Prospective Analysis of the Costs and Benefits of the Clean Air Act under Section 812 of the Act, a panel of outside experts—the Health Effects Subcommittee (HES) of the Advisory Council on Clean Air Compliance Analysis (Council)⁴—provided a limited⁵ and preliminary review of the methodology and

⁴ The Council is an advisory committee with an independent statutory charter that is organized and supported under EPA's SAB.

⁵ Council/HES report: "...in view of the fact that the pilot project is well-underway, the experts have already been selected, and many (if not all) of the interviews have been conducted, the HES sees little potential benefit in providing detailed suggestions about the design or conduct of the pilot study" (EPA-SAB-COUNCIL-ADV-04-002, March 2004, page 34).

design of the expert elicitation (EPA-SAB-COUNCIL-ADV-04-002, March 2004), and provided the following comments:

- "We applaud the Agency's interest in exploring the use of formal expert judgment as a tool for improving uncertainty analysis and believe that the proposed pilot study has great potential to yield important insights. The pilot is well designed to inform subsequent and more comprehensive expert elicitation projects, but relies on the opinions of a relatively small group of experts. It may provide preliminary information about the general magnitude of the mortality effects, and may yield a sense of both the uncertainty inherent in these estimates and the factors largely responsible for such uncertainty. However, until the pilot study methods and results have been subjected to peer review, it may be unwise for the Agency to rely directly on these preliminary results in key policy decisions."
- In presenting results of the pilot elicitation, "the HES advises EPA to present the entire collection of individual judgments; to carefully examine the collection of individual judgments noting the extent of agreement or disagreement; to thoughtfully assess the reasons for any disagreement; and to consider formal combinations of judgments only after such deliberation and with full awareness of the context ..."
- "The HES recognizes that in order to make the pilot tractable it was necessary to limit participation, and is aware of the many factors which must be balanced in the selection of expert panels (Hawkins and Graham, 1988), but is concerned about whether the judgments of such a limited group can reasonably be interpreted as representing a fair and balanced view of the current state of knowledge."

The Council, however, did not provide a peer review of the final report, or the interpretation and application of results. The protocol was then tested on PM scientists from within EPA and external to the Agency, who would not be part of the final elicitation process.

Upon completion of the final report, the pilot was reviewed in a third context. The EPA commissioned a peer review of the pilot from a panel of four experts on the topics of expert elicitation, decision analysis, and uncertainty characterization⁶ (Mansfield, 2004). The review was generally positive. Overall, the reviewers indicated approval of the procedures EPA followed to conduct the expert elicitation, commenting that the procedures "were well documented and followed the standard elicitation protocol." Reviewers cited the approach for selecting experts as a strength of the assessment. They also provided a variety of specific suggestions, including:

⁶ A full report of the peer review is available at www.epa.gov/ttn/ecas/benefits.html.

- Some commented that the number of experts included in the elicitation was small and could possibly be expanded in the future; however, there is not an established method to determine the appropriate number of experts.
- Several reviewers discussed was the lack of a pre- and post-elicitation
  workshop with the participating experts, which is typically conducted in other
  elicitations. They urged EPA to allow sufficient time to include these steps in
  future elicitations but recognized the need to exclude these steps in the pilot.
  Specifically, the reviewers stated that the experts should have communicated
  with each other before and/or after the individual interviews. Group
  communication prior to the individual interviews would have aided in the
  motivation and conditioning steps of the elicitation, while communication,
  either in person or through a summary document, would have allowed an
  expert to adjust his response based on the responses of the other experts.
  - The encoding process of elicitation could be improved. In the elicitation process, the reviewers interpreted that some of the experts provided judgments based on a central tendency before providing judgments on extreme values (upper and lower ranges). This type of sequencing may introduce anchoring or adjustment heuristics, which are associated with biased estimates of uncertainty. Although the authors of the elicitation report introduced the topic of heuristics for the experts, reviewers felt that a more substantive discussion on how the study addressed known sources and any other potential forms of bias was necessary.
- Reviewers also provided considerable comment on whether results of the pilot should be combined into a single estimate. Several of the reviewers preferred that the expert opinions not be combined or stated that they knew of no agreed-upon method for combining results from expert elicitations. This allows for the differences in the individual distributions to be recognized. Two of the reviewers indicated that they were reasonably comfortable with the method used in this study to combine the results, while the other two reviewers offered comments on the combined result of the elicitation. One reviewer stated that the combined distributions do not adequately capture the opinions of individual experts but rather average them out. It is possible in such cases that the combined judgments may generate results that none of the experts could agree on. Another reviewer stated that expert elicitation studies typically do not combine judgments, but if one were to combine them, he

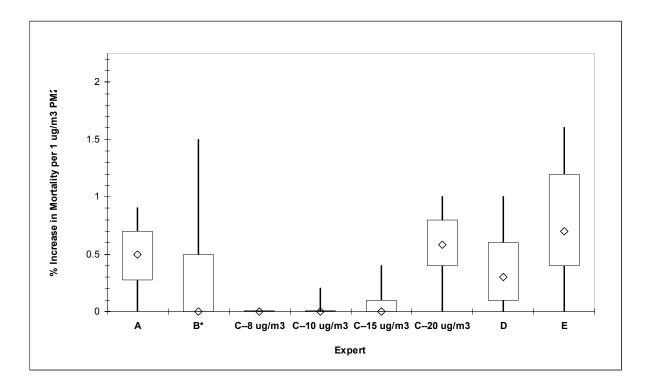
recommended that the response of each be maintained independently from the other experts and run through the benefits model completely prior to combining the results.

#### **B.3.2** Elicitation Results

Figure B-1 displays the responses of the experts to the quantitative elicitation question for the mortality effects of changes in long-term  $PM_{2.5}$  exposures. The distributions provided by each expert, identified by the letters A through E, are depicted as box plots with the diamond symbol showing the median (50th percentile), a circle symbol showing the mean estimate, the box defining the interquartile range (bounded by the 25th and 75th percentiles), and the whiskers defining each expert's 90 percent confidence interval (bounded by the 5th and 95th percentiles of the distribution).

As illustrated by the figures, the experts exhibited considerable variation in both the median values they reported and in the spread of uncertainty about the median. In response to the question concerning the effects of changes in long-term exposures to  $PM_{2.5}$ , the median value ranged from values at or near zero to a 0.7 percent increase in annual nonaccidental mortality per 1 µg/m³ increase in annual mean  $PM_{2.5}$  concentration (within a range of  $PM_{2.5}$  concentrations from 8 to 20 µg/m³). The variation in the responses for the effects of long-term exposures largely reflects differences of opinion among the experts on a number of factors such as the interpretation of key epidemiological results from long-term cohort studies, the likelihood of a causal relationship, and the shape of the C-R function. Some observations concerning the outcome of the individual expert judgments are provided below:

*Key Cohort Studies.* The experts' nonzero responses for the percentage change in annual mortality were mostly influenced by the Krewski et al. (2000) reanalysis of the original American Cancer Society (ACS) cohort study and by the later Pope et al. (2002) update of the ACS study that included additional years of follow-up. In the characterization of uncertainty upper bounds, none of the experts placed substantial weight on the mortality estimates from the Six-Cities study (Dockery et al., 1993) in composing their quantitative responses, despite citing numerous strengths of that analysis. Concern about sample size and representativeness of the Six-Cities study for the entire United States appeared to be major reasons for de-emphasizing those results. In addition, all of the experts gave a value of zero at the 5th percentile, and thus the C-R functions are bounded by zero. We use a normal distribution to characterize the pilot results, but the distribution could potentially be skewed because of the bounding at zero.



## Figure B-1. Summary of Experts' Judgments About the Percentage Increase in Annual Average Nonaccidental Mortality Associated with a 1 µg/m³ Increase in Annual Average Exposures to PM_{2.5}

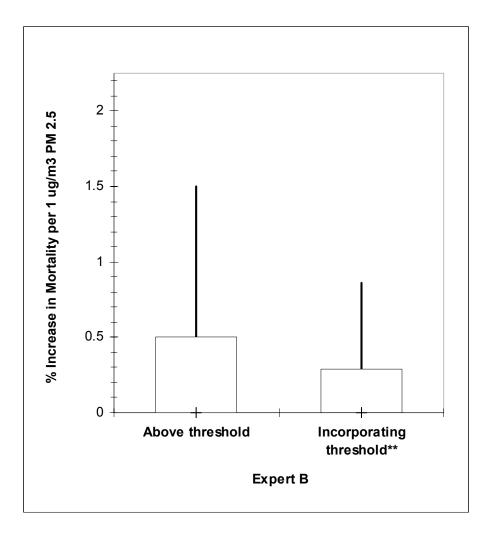
- * Expert B specified this distribution for the PM/mortality coefficient above an uncertain threshold that he characterized as ranging between 4 and 15 with a modal value of  $12 \mu g/m^3$ . As illustrated here, considerable variation exists in both the median values and the spread of uncertainty provided by the experts. The median value of the percentage change in annual nonaccidental mortality per unit change in annual PM_{2.5} concentrations from 8 to  $20 \mu g/m^3$ ) ranged from values at or near zero to a value of 0.7 percent. The variation in the responses largely reflects differences in the amount of uncertainty each expert considered inherent in the key epidemiological results from long-term cohort studies, the likelihood of a causal relationship, and the shape of the C-R function. The technical report (IEc, 2004) provides detailed descriptions of the experts' judgments about these factors, but we present a few brief observations relative to their responses below.
- ** Expert C specified a nonlinear model and provided distributions for the slope of the curve at four discrete concentrations within the range.

*Causality for Long-Term Effects.* Three of the five experts gave distributions more heavily weighted towards zero. Those experts were also the ones who expressed the lowest probability of a causal effect of long-term exposure to  $PM_{2.5}$  in response to the preliminary questions. All of the experts placed at least a 5 percent probability on the possibility that there is no causal relationship between fine PM exposure and mortality; as a result, all experts gave a fifth percentile value for the C-R coefficient of zero. For most of the experts, this was based primarily on residual concerns about the strength of the mechanistic link between the exposures and mortality.

Shape of the C-R Function for Long-Term Effects. The other key determinant of each expert's responses for long-term effects was his assumption about the nature of the C-R function across the range of baseline annual average  $PM_{2.5}$  concentrations assumed in the pilot (8 to 20 µg/m³). Three experts (A, D, and E) assumed that the function relating mortality with PM concentrations would be log-linear with constant slope over the specified range. They therefore gave a single estimate of the distribution of the slope describing that log-linear function. The other two experts provided more complex responses.

Expert B assumed a population threshold in his model, below which there would be no effect of increased  $PM_{2.5}$  exposure and above which the relationship would be log-linear. He characterized his estimate of a possible threshold as uncertain, ranging between 4 µg/m³ and 15 µg/m³, with a modal value of 12 µg/m³. He then described a distribution for the slope for the log-linear function that might exist above the threshold; this distribution is depicted in Figure B-2. The effect of incorporating the uncertain threshold is essentially to shift his entire distribution downward.

Expert C believed that the increased relative risks for mortality observed in the cohort studies were likely to be the result of exposures at the higher end of the exposure range, and he expected there to be a declining effect on mortality with decreasing levels of  $PM_{2.5}$ . He also argued that some practical concentration threshold was likely to exist below which we would not observe any increase in mortality. He reflected these beliefs by developing a nonlinear model within the range from 8 to 20 µg/m³; he described the model by providing distributions for the slope of the curve at four discrete concentrations within the range.



# Figure B-2. Expert B's Distributions for the Percentage Increase in Annual Nonaccidental Mortality Associated with a $1 \mu g/m^3$ Increase in Long-Term Exposures to PM_{2.5}: Comparison of His Distribution Above a Threshold to His Expected Distribution* for the Range 8-20 $\mu g/m^3$

* Expert B specified the threshold as uncertain between 4 and 15 µg/m³ with a modal value at 12 µg/m³. He assumed the percentage increase in mortality to increase linearly with concentration above the threshold. His effective distribution was simulated using Monte Carlo techniques assuming an underlying distribution of population-weighted annual average PM_{2.5} concentrations for the United States generated from the BenMAP model (see the technical report [IEc, 2004] for details).

#### **B.3.3** Experts' Views of Sources of Uncertainty

The experts were asked at several points during the interview to discuss the key sources of potential bias and uncertainty in current evidence on which they relied for their judgments. In the context of the quantitative discussion they were asked to list the top five issues. They were encouraged to think about how these issues would affect the uncertainty surrounding their best estimate of the potential impact on total mortality of a small change in long-term exposure to  $PM_{2.5}$ . The tables summarizing the factors identified by each expert may be found in Appendix E of the technical report (IEc, 2004).

Many of the same factors appeared in the list of the five experts. However, the experts often differed on whether a particular factor was a source of potential bias or uncertainty. Some of the common concerns raised as either sources of bias or uncertainty were

- residual confounding by smoking,
- residual confounding by "lifestyle" or other personal factors or "stressors,"
- exposure errors/misclassification,
- the role of co-pollutants as confounders or effect modifiers,
- impact of the relative toxicity of PM components,
- representativeness of the cohort populations with respect to the general U.S. population, and
- investigator/publication biases.

Despite the many qualitative discussions about sources of uncertainty, because the pilot study did not elicit quantitative judgments about the size and nature of impacts of each source of uncertainty and bias, we were unable to systematically evaluate the nature of the influence of these factors on the quantitative results provided by each expert unless an expert explicitly adjusted his estimates by a particular factor.

#### **B.3.5** Limitations in Pilot Elicitation Design

The pilot elicitation has afforded many opportunities for learning about expert elicitation in the context of economic benefits analysis. During the process of developing the protocol, the project team noted limitations inherent in the design of the pilot, some of which were also commented on by the SAB/HES in their review of the protocol prior to conducting the elicitation. Additional detail on the strengths and weaknesses of the pilot are provided in the technical report (IEc, 2004).

The limited scope of the pilot meant that a full expert elicitation process was truncated, and many aspects of the uncertainty surrounding the  $PM_{2.5}$ /mortality relationship could not be characterized quantitatively. Recognizing this, the results of the pilot are only used in this benefits estimation for illustrative purposes.

• Small panel of experts—Because of resource constraints we limited the pilot to a panel of five experts. As noted above, the SAB-HES (in Section B.3.1) brings into question whether the judgments of such a limited group can reasonably be interpreted as representing a fair and balanced view of the current state of knowledge.

Little analytical research has been conducted on the more difficult question of how to determine the ideal number of experts for a particular application. We have not found any analyses of the effect of expert panel size based on comparisons of empirical results of expert judgment studies. A theoretical analysis by Clemen and Winkler (1985) suggests that where data sources are moderately positively dependent there are diminishing marginal returns to the value of information associated with each additional data source. In the context of expert judgment studies, such a result implies that when dealing with experts of similar backgrounds who rely on the same models and studies, a larger expert panel may not provide significantly higher quality results than a smaller one. However, the addition of an expert expected to provide a more independent assessment, such as an expert from a different but pertinent field, would be expected to exhibit a much greater value of information. Clemen and Winkler (1999) note that "heterogeneity among experts is highly desirable." These findings would appear to support addressing complex issues using a panel comprising relatively small subgroups (perhaps three to five experts each) from multiple disciplines. Although the decision analysis field tends to use relatively small sample sizes (i.e., typically 5 to 10 experts), some are not comfortable with obtaining a combined distribution from such small numbers in the absence of an a priori assessment of the degree to which the expert panel is likely to be representative of the overall population of relevant experts on the question of interest.

• Use of an aggregate elicitation question—The expert judgment literature discusses two broad approaches to elicitation of judgments: an aggregated and a disaggregated approach. As the term implies, an aggregated approach asks the expert to estimate the quantity of interest directly, for example, the numbers of newspapers sold in the United States in a particular year. In a disaggregated approach, the expert (or group of experts) is asked to construct a model for estimating the quantity of interest and is asked directly about the inputs to that

model (e.g., population in each state, percentage of the population that reads newspapers). The intuition is that it is easier for experts to answer questions about the intermediate quantities than about the total quantity.

The project team carefully considered the relative advantages and disadvantages of the two approaches, however, the time and resources necessary to develop a disaggregated model structure drove the decision to undertake an aggregate approach to elicit the C-R coefficient for the  $PM_{2.5}$ /mortality relationship. Based on advice from the SAB-HES, the project team felt that separate questions to address effects of short- and long-term exposures, though still at a high level of aggregation, would prove to be easier for experts to address than a question that "rolled up" all the effects into a single estimate. This level of disaggregation also enabled the elicitation team to explore with experts possible overlap in reported mortality effects detected using short-term and long-term epidemiological studies. Nonetheless, a major goal of the preliminary and follow-up questions in the protocol was to identify critical issues that could be addressed by developing a more disaggregated approach in a future assessment.

The aggregated design limits our ability to determine the influence of any one key factor over others in a large list of issues that the experts were to consider prior to answering the quantitative question. It also limited the ability of the experts to express their views about the difference in the C-R function based on the location in the United States (i.e., the demographics of the exposed population, the air concentration of PM and/or PM mixture).

- No workshop was conducted—It is customary to conduct a workshop prior to the elicitation interview with the experts. This allows the experts to become familiar with the protocol and to discuss methods to limit bias during the interview. Because of time constraints for the pilot, we did not conduct a pre-elicitation workshop.
- No calibration of experts—In some elicitation studies, the authors use a calibration measure to weight the experts appropriately for the purposes of combining the results of the elicitation. We do not have calibration measures that could be used to assess the results of this pilot. At this point, we can only assess the process—did the pilot assessment employ a structure, supporting materials, and a process that enabled experts to make judgments that would be likely to be well calibrated? Without calibration measures, we cannot weight experts based on their performance on calibration and thus, we present only an equal weighting of the responses. The peer reviewers agree that if a combination is provided, then the equal weighting of judgments is preferred.

#### **B.3.6** Combining the Expert Judgments for Application to Economic Benefit Analyses

Many methods are available to combine the responses from the experts. Each method has advantages and disadvantages from a statistical viewpoint. The project team is not aware of any rule of thumb in statistics that would provide guidance for combining linear and nonlinear functions. Therefore, we considered four alternative methods for combining the results as an illustration of potential combinations of the results. The peer review provides extensive comments on whether a combination is necessary and on the methods employed here.

Analysts must give careful thought to whether and how to combine the results of individual expert judgments into a single distribution. When dealing with a limited number of experts, the analyst must be particularly careful to identify the influence of each expert's response on the combined distribution. Therefore, we considered four alternative methods for combining the pilot results, each of which had limitations. The peer review also provides a lengthy discussion of issues in combining expert judgments. In this section, we discuss the issues we considered in combining the results of the pilot and how we came to the conclusion that for the illustrative benefits analysis presented in Section B.5 below, we would present both the individual quantitative distributions of the C-R coefficient elicited from the five experts interviewed and results based on a probabilistic estimate that represents the combined results of the pilot based on an equal weighting of the calculated change in mortality incidence based on the individual judgments.

#### B.3.6.1 Background

Some investigators (e.g., Hawkins and Graham [1990]; Winkler and Wallsten [1995]; and Morgan et al. [1984]) have preferred to keep expert opinions separate to preserve the diversity of opinion on the issues of interest. In such situations, presenting a range of values expressed by the experts can help decision makers to understand the sensitivity of their analyses to the analytical model chosen, thereby bounding possible outcomes. Individual judgments also can illustrate varying opinions arising from different disciplinary perspectives or from the rational selection of alternative theoretical models or data sets (Morgan and Henrion, 1990). Nonetheless, analysts are often interested in developing a single distribution of values that reflects a synthesis of the judgments elicited from a group of experts.

On the other hand, there are advantages to combining the results across experts. An extensive literature describes methods for combining expert judgments. These methods can be broadly classified as either mathematical or behavioral (Clemen and Winkler, 1999).

Mathematical approaches range from simple averaging of responses to much more complex models incorporating information about the quality of expert responses, potential dependence among expert judgments, or (in the case of Bayesian methods) prior probability distributions about the variable of interest. Behavioral approaches require the interaction of experts in an effort to encourage them to achieve consensus, either through face-to-face meetings or through the exchange of information about judgments among experts. As noted in the technical report (IEc, 2004), analysts have raised both methodological and practical concerns with respect to the behavioral approach. Therefore, we used a mathematical combination process to derive a single distribution.

One advantage of mathematical combination over behavioral approaches is the ability to be completely transparent about how weights have been assigned to the judgments of specific experts and about what assumptions have been made concerning the degree of correlation between experts. Several approaches can be used to assign weights to individual experts. Weights can be assigned based on the analyst's opinion of the relative expertise of each expert; on a quantitative assessment of the calibration and informativeness (i.e., precision) of each expert as determined from their responses to a set of calibration questions (as described in Cooke [1991]); or on weights assigned by each expert, either to him or herself or to the other experts on the panel (see Evans et al. [1994b] for an example of this approach). Ideally, such a weighting system would address problems of uneven calibration and informativeness across experts, as well as potential motivational biases (Cooke, 1991).⁷

At the design stages of the pilot, we decided that the resulting expert judgments would be combined using equal weights, essentially calculating the arithmetic mean of the expert responses, for simplicity and transparency. Some empirical evidence suggests that the simple combination rules, like equal weighting, perform equally well when compared to more complex methods in terms of calibration scores for the combined results (Clemen and Winkler, 1999).

#### **B.3.6.2** Alternative Combination Methods

Although a combination method using equal weights for the results of each expert is straightforward in principle, applying it in this context of the results of the pilot was complicated by the fact that the elicitation protocol gave the experts freedom to specify different forms for the C-R function. If all the experts had chosen the same form of the C-R

⁷ "Motivational bias" refers to the willful distortion of an expert's true judgments. The origins of this bias can vary but could include, for example, a reluctance to contradict views expressed by one's employer or a deliberate attempt to skew the outcome of the study for political gain.

function (e.g., if each expert had specified a log-linear C-R function with a constant, but uncertain, C-R coefficient [i.e., slope] over the PM range specified in the protocol), the combination of their distributions for the C-R coefficient would require a simple averaging across experts at each elicited percentile. However, in this assessment, three experts specified log-linear functions with constant C-R coefficients over the specified range of  $PM_{2.5}$  concentrations, and two of the experts specified the C-R coefficient was likely to vary over the range of specified  $PM_{2.5}$  concentrations (as discussed in Section B.4.2 above). These more complex C-R functions necessitated some additional steps in calculating the combined results.

As discussed in the technical report for the pilot (IEc, 2004), individual responses either can be combined before application of the benefits model or during the application of the model, allowing each expert's C-R function to be estimated in the benefits model independently. We considered three alternative approaches to combining the expert judgments before application to the benefits model each of which differs in how the combined estimate accounts for the underlying particulate air pollution levels. We considered the use of (1) a uniform distribution and equal weighting, which involves taking a simple average of the responses across experts for each percentile, (2) a normal distribution describing the population-weighted annual average  $PM_{2.5}$  concentration data, and (3) a combined distribution specified at four intervals of  $PM_{2.5}$  concentrations that coincide with those specified by Expert B.

Overall, the combination methods considered result in fairly similar results at the median and mean relative risk estimate. However, slight differences occur in the tails of the distribution in their characterization of uncertainty. In particular, when combining the upper bound is averaged out to a lower value that may skew the results (in comparison to the views expressed by the experts). Thus, the resulting estimates from the combined distribution may not be estimates to which the experts would agree (i.e, Expert E may not agree with a lowering of the upper bound estimate due to averaging across experts). In Figure B-3, the C-R function for the population-weighted combination method was compared to the existing cohort epidemiological studies of the long-term  $PM_{2.5}$ /mortality relationship. We observe that the results of the pilot elicitation are generally within the range of findings from these epidemiological studies. However, as expected, the elicitation results in a larger spread of uncertainty than is given by the standard errors of the individual studies.

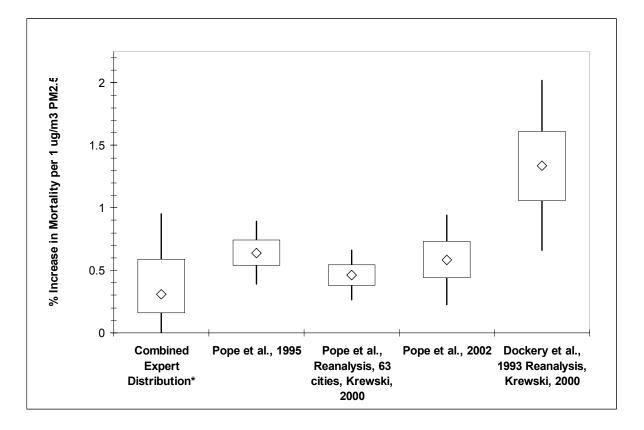


Figure B-3. Comparison of A Combination of the Distributions of Expert Judgment to Selected Published Studies

For the purposes of providing a supplemental analysis of the CAIR, we decided to combine the results of the pilot after applying each experts' distribution independently through BenMAP, the model EPA currently uses for economic benefits analyses of air quality regulations affecting PM and other criteria pollutants. Specifically, we derived the total mortality incidence for each expert and combined (or pooled) the estimates into an aggregate value before taking an average of the mortality incidence. This is referred to as a "pooled" approach and is used in our modeling framework for other benefits endpoints that have multiple C-R functions (due to multiple studies). We prefer the pooled approach because it seems to reduce the amount of alteration of the actual step-function responses

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provided by Experts B and C (although some adjustments must still be made).⁸ Details of the illustration are provided in Section B.6.

#### **B.4** Illustrative Application of Pilot Expert Elicitation Results

In this section, we illustrate how expert judgments can be applied in a benefits analysis. We estimated avoided incidence of mortality associated with the CAIR rule for each expert and also applied the pooled approach for combining results across participants to develop a combined distribution of avoided incidence of premature mortality. We then used Monte Carlo simulations to combine the expert judgment-based distributions with the distribution for VSL and with the values for other health and welfare endpoints. We present the resulting distributions of total dollar benefits to demonstrate how using expert elicitation can improve the characterization of the overall uncertainty in benefits estimates. The values generated below are not intended to replace the primary estimate of benefits of CAIR. They are included solely as an illustration of expert elicitation-based distributions that characterize the uncertainty of the estimate of premature mortality associated with long-term exposure to  $PM_{2.5}$  rather than a data-derived distribution.

#### B.4.1 Method

#### B.4.1.1 C-R Distribution Based on Combined Results Across Experts

As discussed in Section B.4.5, we converted each expert's percentile responses about mortality associated with long-term exposure into a custom distribution such that each percentile is correctly represented and percentiles in between are represented as continuous functions (custom distributions were generated using Crystal Ball and are represented as 15,000 equally probable points).

For Experts A, D, and E, we used a standard log-linear functional form:

$$\Delta y = y_0 \cdot (e^{\beta \cdot \Delta x} - 1), \qquad (B.5)$$

⁸ Expert B specified a distribution for the C-R coefficient for PM_{2.5} concentrations above a threshold and assigned the coefficient a value of zero for all PM concentrations below the threshold. He then specified a probability distribution to describe the uncertainty about the threshold value. Expert C specified separate distributions for the C-R coefficient at four discrete points within the concentration ranges defined in the protocol to represent a continuous C-R function whose slope varied with the PM_{2.5} concentration. Expert C indicated that the coefficient value between these points was best modeled as a continuous function, rather than a step function. Both experts assumed the same functional forms in responding to elicitation question.

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where we set  $\beta$  equal to ln(1+B/100), where B is the percentage change in all-cause mortality associated with a 1 µg reduction in PM_{2.5}. BenMAP then represents the distribution of  $\Delta y$  based on the custom distribution of  $\beta$ .

The conditional functions of Experts B and C required us to estimate some values on the C-R function between the points that were elicited, which requires an extrapolation from the response provided in the pilot to create continuous distributions. This can alter the true response given by these experts to the elicitation. This component of the combination was not included in the peer-review material.

Specifically, Expert C provided a set of conditional C-R functions for different baseline levels of  $PM_{2.5}$ . Expert C provided four conditional responses, one for 8 µg/m³, one for 10 µg/m³, one for 15 µg/m³, and one for 20 µg/m³. To "fill in" the C-R function for intermediate baseline  $PM_{2.5}$  values, we linearly interpolated between the responses for each pair of points (e.g., 10 to 15 or 15 to 20). We calculated interpolated values for 13 points, ranging from 8 µg to 20 µg. For baseline values less than 8 µg, we assigned a value of zero (essentially assuming a threshold at 8 µg). For baseline values greater than 20 µg, we assigned the values provided by Expert C for 20 µg. This may result in an underestimate of the incidence of mortality for Expert C. For each of the conditional functions, we used a loglinear specification, similar to A, D, and E. Total incidence of mortality for Expert C is the sum of the conditional estimates over the range of baseline air concentrations.

Expert B provided a log-linear C-R function, conditional on an unknown threshold characterized by a triangular distribution bounded by 4  $\mu$ g and 15  $\mu$ g, with a mode at 12  $\mu$ g. We discretized the triangular distribution into 12 ranges of unit length (e.g., 4 to 5, 5 to 6) and calculated the expected value of the response at each population grid cell based on the observed baseline PM_{2.5} and the probability of that baseline value exceeding the potential threshold. We assumed that if a grid cell has a baseline value above the threshold, then the full value of the reduction in PM_{2.5} at that grid cell is associated with a reduction in mortality. This may result in an overestimate of the mortality impact for Expert B because for grid cells where the baseline level is only marginally above the threshold, a benefit might only accrue to the change in PM_{2.5} down to the threshold. The rest of the change would not result in any mortality reduction. Because most of the changes in air quality are relatively small (population-weighted change in annual mean PM_{2.5} is around 1  $\mu$ g), and larger changes tend to occur where there are higher baseline PM_{2.5} levels, this should not be a large issue.

To provide context for the estimates based on the experts providing conditional and threshold specifications, it is useful to summarize the CAIR baseline  $PM_{2.5}$  levels in 2015. Table B-5 lists the population distribution of baseline concentrations of  $PM_{2.5}$  in 2015.

Baseline PM _{2.5} (µg/m ³ )	2015 Population (millions)	Percent of Total 2015 Population
PM _{2.5} <5	4.3	1.3%
$5 \le PM_{2.5} \le 10$	81.2	25.6%
$10 \le PM_{2.5} \le 15$	190.5	60.1%
$15 \le PM_{2.5} \le 20$	29.2	9.2%
$20 \le PM_{2.5} \le 25$	11.0	3.5%
$25 \le PM_{2.5} \le 30$	0.7	0.2%

 Table B-5. Population Distribution of Baseline Ambient PM2.5

#### B.4.1.2 Estimated Reduction in Premature Morality and Valuation

Based on the air quality modeling conducted for the final CAIR, we calculated the reduction in incidence of premature mortality associated with  $PM_{2.5}$  and the value of that reduction. We used Monte Carlo simulations to derive the distributions of the dollar values of estimated reductions in premature mortality. For each expert, the Monte Carlo simulation generates a dollar value by randomly sampling from the distribution of the reduction in mortality incidence and the distribution of VSL (normally distributed with a mean of \$5.5 and a 95 percent confidence interval between \$1 and \$10 million) and multiplying the values together. This yields an estimate of the dollar value of the mortality reductions. This process is repeated 5,000 times to generate a distribution for each expert individually and for the combined (pooled) distribution, as well as for the distribution derived from the Pope et al. (2002) study.

#### **B.4.2** Results

Figure B-4 presents box plots that display the distribution of the reduction in  $PM_{2.5}$ -related premature mortality based on the C-R distributions provided by each expert, as well as a pooled distribution across the experts.⁹ The estimated distributions of the reduction in  $PM_{2.5}$ -related premature mortality corresponding to each individual expert's reported distribution (labeled A through E) of percent risk are presented in Figure B-4, along with a

⁹ As discussed above, the elicitation results were combined assuming equal weight for each expert's distribution. We assumed complete dependence of the expert's distributions for this illustrative analysis, so that each percentile of the pooled distribution is simply the average of the corresponding percentiles of the five experts.

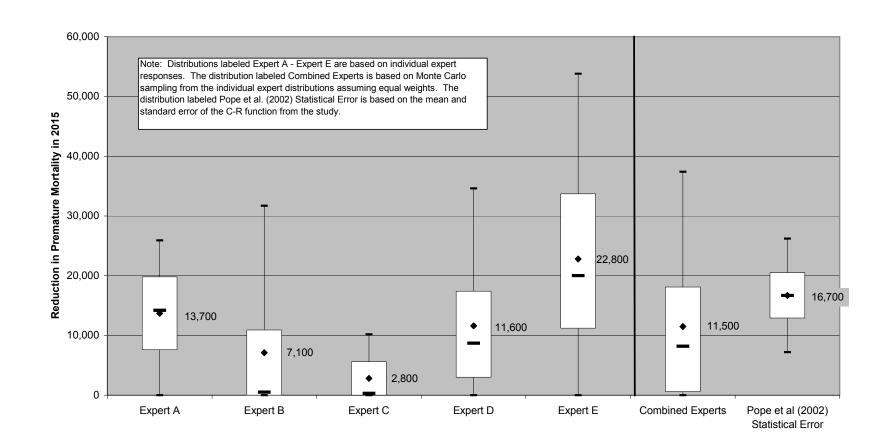


Figure B-4. Results of Illustrative Application of Pilot Expert Elicitation: Annual Reductions in Premature Mortality in 2015 Associated with the Clean Air Interstate Rule

pooled estimate assuming equal weight for each expert's distribution and the Pope et al. (2002) results for comparison. Corresponding distributions of the dollar value of the reductions in premature mortality are presented in Figure B-6. The distributions are depicted as box plots with the diamond symbol ( $\blacklozenge$ ) showing the mean, the dash (–) showing the median (50th percentile), the box defining the interquartile range (bounded by the 25th and 75th percentiles), and the whiskers defining the 90 percent confidence interval (bounded by the 5th and 95th percentiles of the distribution). For comparison, the figure also displays the distribution derived from the statistical error associated with Pope et al. (2002).

The figure shows that the <u>average</u> annual number of premature deaths avoided because of CAIR in 2015 ranges from approximately 3,000 to 23,000, depending on the C-R function used. The medians span zero to 20,000, with the zero value due to the low threshold associated with one of the expert's distributions. At the means of the distributions, 45 percent more premature deaths are predicted to be avoided by the estimate based on Pope et al. (2002) than based on the estimate pooled across the five experts. Specifically, because less than a quarter of the population is expected to live in areas with PM_{2.5} levels above the threshold specified by Expert C, and much of the decrease in PM_{2.5} associated with CAIR occurs below that threshold, a much smaller decrease in premature morality is predicted for Expert C than those experts who provided continuous C-R functions down to zero (PM_{2.5}) as well as for Expert B who provided an uncertain threshold. Furthermore, note that above the 50th percentile, the C-R functions provided by all of the experts predict positive benefits from the modeled control option.

The boxplots displayed in Figure B-4 are derived by applying the C-R distributions specified by each expert (as presented in Figure B-1) to the change in air quality predicted by the final CAIR. Although the Figures B-4 and B-1 show similar patterns, there are important differences. Specifically, the ratio of 75th percentiles of the C-R functions specified by Experts A and B (as denoted in Figure B-1) is 0.4, whereas the ratio of the predicted change in incidence of premature mortality associated with the modeled preliminary control option is 0.5. This 25 percent increase (from 0.4 to 0.5) in the ratio highlights the impact of the extent of the predicted air quality change on the choice of C-R function used in the benefits analysis.

The combined expert distribution depicted in Figure B-4 provides additional insights. The combined (average) distribution has a 90 percent credible interval between zero and 37,000. When compared with results derived from the Pope et al. (2002) study, it is clear that the combined expert distribution reflects greater uncertainty about the estimated reduction in premature mortality, which is expected given that the elicitation exercise was designed to encompass more sources of uncertainty than were addressed in the Pope et al. study, including fundamental model uncertainty. In addition, the expert judgments place more weight on the lower end of the distribution than the Pope et al. study. The mean estimate from the combined expert distribution is over 30 percent lower than the mean

derived from the Pope et al. (2002) distribution. However, the 90 percent confidence interval based on the standard error from Pope et al. (2002) is completely contained within the 90 percent credible interval of the combined expert distribution.

Figure B-5 shows the same data using cumulative distribution functions (CDFs). This figure is valuable for demonstrating differences in degree of certainty in achieving specific reductions in premature mortality. For instance, the Pope et al. (2002) C-R distribution predicts a 90 percent chance that there will be more than 10,000 fewer premature deaths, whereas the pooled distribution from the pilot predicts less than a 50 percent chance of more than 10,000 fewer premature deaths resulting from CAIR. The probabilities associated with the individual experts for avoiding 10,000 or more premature deaths range from about 5 percent to 80 percent, demonstrating once again the sensitivity of the estimate to assumptions regarding the C-R function. The CDFs of the estimated reductions in premature mortality show that for several experts there is a small probability of a substantially more reduction in pre-mature mortality. For example, the 75th percentile of the distribution based on Expert B's responses is at 11,000 fewer deaths, while the 98th percentile for that distribution is over three times higher, at 35,000. The CDF also shows that, although most of the experts provided fairly wide distributions reflecting incorporation of information beyond what is demonstrated in any one empirical study, the CDF based on Expert C's responses is much narrower, reflecting the high degree of confidence he placed on the existence of a threshold below 15 µg.

Figures B-6 and B-7 use box plots and CDFs to display the estimated dollar value of these annual reductions in premature mortality. Whereas the average based on the Pope et al. (2002) distribution is \$93 billion, the average based on the pooled estimate from the pilot is \$64 billion, a difference of approximately one-third. Once the C-R distributions are combined with the VSL distributions, not only are the mean values closer to one another, but the distributions show considerably more overlap.

Because these distributions are the result of a Monte Carlo simulation combining the non-normal distributions for reductions in mortality with a normal distribution for VSL, the resulting distributions will also be non-normal, but the shape depends on the skewness of each of the input distribution of mortality reductions. For example, the ratio of the 95th to 75th percentile of mortality reductions for Expert B is 2.9, while the same ratio for the value of mortality reductions is 3.4, indicating the value distribution is more skewed than the reductions distribution. In general, combining normal or left-skewed distributions in a multiplicative fashion will result in left-skewed distributions with greater skewness than the input distributions. So even for the normally distributed estimates based on Pope et al. (2002), the value distribution is somewhat skewed, because it is the result of multiplying two normally distributed random variables.

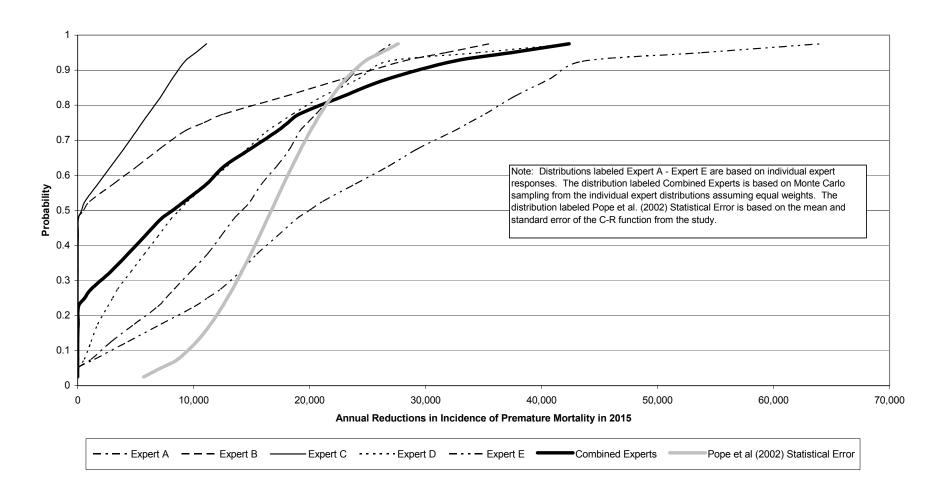


Figure B-5. Cumulative Distribution Functions for Annual Reductions in Premature Mortality in 2015 Associated with the Clean Air Interstate Rule

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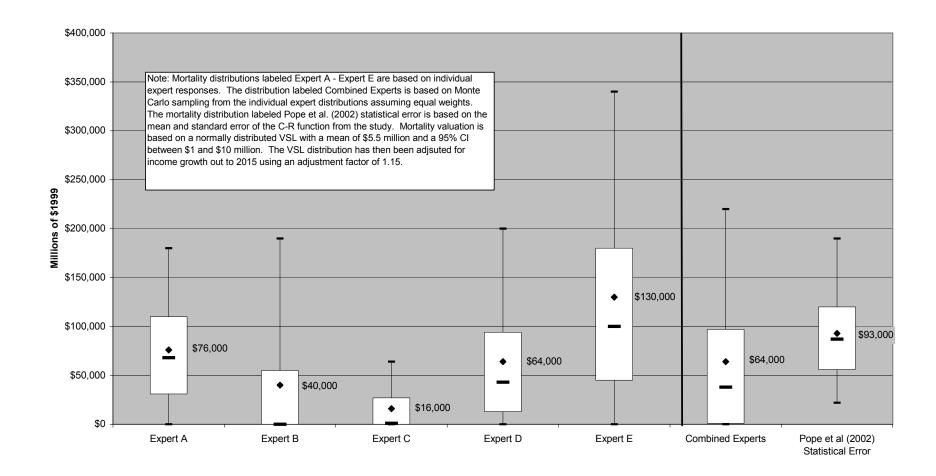


Figure B-6. Results of Illustrative Application of Pilot Expert Elicitation: Dollar Value of Annual Reductions in Premature Mortality in 2015 Associated with the Clean Air Interstate Rule

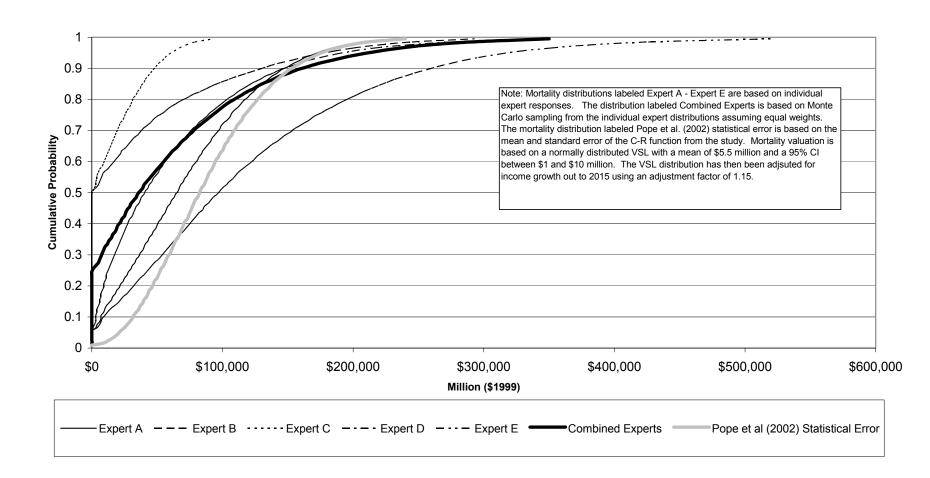


Figure B-7. Cumulative Distribution Functions for Dollar Value of Annual Reductions in Premature Mortality in 2015 Associated with the Clean Air Interstate Rule

The shapes of the two distributions are more similar in this case because both reflect the same additional information in the VSL distribution. This demonstrates that, as additional sources of uncertainty are added to the analysis, the influence of any single source of uncertainty will fall. Because VSL is a large source of uncertainty, the influence on overall uncertainty relative to the distribution of the mortality reduction is also large. All of the distributions of the value of mortality reductions have a small negative tail, this time because of propagation of the normally distributed VSL, which has a small amount of the distribution below zero. Again, we interpret this as a statistical artifact rather than a true probability that the value of a statistical life is negative (implying that individuals would pay to increase the risk of death).

We used additional Monte Carlo simulations to combine the expert-based distributions for the dollar benefits of mortality with the distributions of dollar benefits for the remaining health and welfare endpoints to derive estimates of the overall distribution of total dollar benefits.¹⁰ The box plots for these distributions of overall dollar benefits associated with CAIR in 2015 are presented in Figure B-8. Because mortality accounts for over 90 percent of the benefits, the addition of other endpoints has little impact on the overall distributions. The overall mean annual total dollar benefits in 2015 for the distribution incorporating the combined expert distribution for reductions in premature mortality is \$74 billion, compared to \$100 billion for the results derived from the Pope et al. (2002) study.

The CDFs for total dollar benefits are provided in Figure B-9. These again suggest that using the pilot expert elicitation-based representation of uncertainty in the relationship between  $PM_{2.5}$  and premature mortality has a large impact on the shape and range of the distribution of total benefits. The Pope et al. (2002) derived results have an approximately Weibull-shaped distribution with a range from 5th to 95th percentiles of \$26 billion to \$210 billion, or about one order of magnitude. The distribution of total dollar benefits incorporating the combined expert distribution for reductions in premature mortality has a much more skewed shape with an elongated positive tail above the 75th percentile with a range from 5th to 95th percentiles of \$3 billion to \$240 billion, or about two orders of magnitude, with significant probability mass at the lower end of the range.

¹⁰Note that visibility benefits are treated as fixed for this illustrative analysis. We are working on methods to characterize the uncertainty in visibility and other nonhealth benefits.

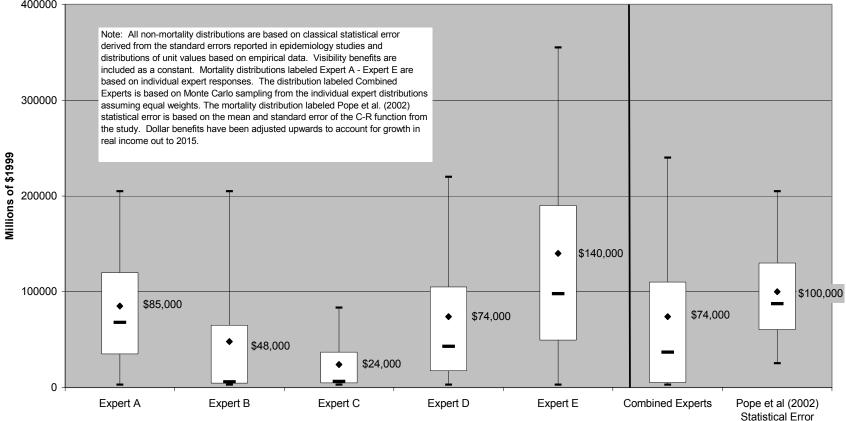


Figure B-8. Results of Illustrative Application of Pilot Expert Elicitation: Dollar Value of Total Annual Benefits in 2015 Associated with the Clean Air Interstate Rule

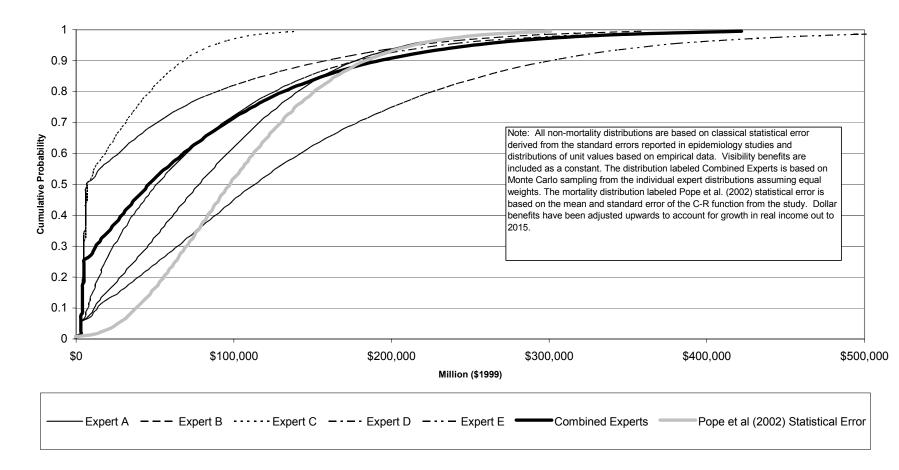


Figure B-9. Cumulative Distribution Functions of Dollar Value of Total Annual PM-Related Health and Visibility Benefits in 2015 Associated with the Clean Air Interstate Rule

#### **B.5.3** Limitations of the Application of the Pilot Elicitation Results to the CAIR Scenario

The results presented in this section should be viewed cautiously given the limited scope of the pilot and the limitations of the elicitation design and methods used to combine the expert judgments discussed above. Therefore, the results presented above should be considered "illustrative" until the methods used to interpret and apply the results of the pilot have been peer reviewed by EPA's SAB.

Specific limitations of the illustrative application include the following:

- Extrapolation of percentile responses provided by individual experts. Each expert provided minimum and maximum values, as well as the 5th, 25th, 50th, 75th, and 95th percentiles. To generate the continuous distributions of mortality impacts, we had to make assumptions about the continuity of the distributions between the reported percentiles. The use of assumptions adds uncertainty to the results.
- Interpolation of C-R relationship across  $PM_{2.5}$  levels. Expert C provided a set of conditional distributions of the C-R relationship conditioned on the baseline level of  $PM_{2.5}$ . Because he only provided functions for a limited number of baseline levels, we had to interpolate the values between levels, introducing additional uncertainty. In addition, Expert C provided no information on the C-R function for baseline  $PM_{2.5}$  levels below 8  $\mu g/m^3$  or above 20  $\mu g/m^3$ . We assumed no mortality impacts for baseline levels lower than 8 and no increase in the C-R function above 20. If the interpolation method was incorrect, it likely biased our results downward.
- Interpretation of Expert B's results. Expert B provided a conditional distribution for the C-R function, conditioned on an uncertain threshold. Expert B provided additional information about the shape of the distribution for the threshold. To develop an applied function, we assumed that the uncertain threshold could be incorporated into the C-R function by constructing an expected value function. If our interpretation was flawed, the specific functions introduced may lead to a slight overestimate of mortality impacts.
- Ranges based on individual experts should be viewed with caution because they represent only a single individual's interpretation of the state of knowledge about PM and mortality. Results for individual experts should not be extracted and presented without reference to the full range of results across the five experts.
- Any range of results presented based on the application of the pilot results in the CAIR scenario should be presented along with their relative likelihood of occurring (i.e., the percentile represented in the distribution).

#### **B.6** References

- Agency for Healthcare Research and Quality (AHRQ). 2000. HCUPnet, Healthcare Cost and Utilization Project.
- Amaral, D. 1983. Estimating Uncertainty in Policy Analysis: Health Effects from inhaled Sulfur Oxides. Ph.D. Thesis, Department of Engineering and Public Policy, Carnegie Mellon University, Pittsburgh, PA.
- Ayyub, B.M. 2002. *Elicitation of Expert Opinions for Uncertainty and Risks*. CRC Press, Florida.
- Clemen, R.T., and R.L. Winkler. 1999. "Combining Probability Distributions From Experts in Risk Analysis," Risk Analysis. 19: 187-203.

Cooke, R. 1991. Experts in Uncertainty. Oxford University Press, New York.

- Cropper, M.L., and A.J. Krupnick. 1990. "The Social Costs of Chronic Heart and Lung Disease." Resources for the Future. Washington, DC. Discussion Paper QE 89-16-REV.
- Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris, and F.E. Speizer. 1993. "An Association between Air Pollution and Mortality in Six U.S. Cities." *New England Journal of Medicine* 329(24):1753-1759.
- EPA-SAB-COUNCIL-ADV-04-002. March 2004. Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis—Benefits and Costs of the Clean Air Act, 1990-2020: Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis.
- Evans, J.S., J.D. Graham, G.M. Gray, R.L. Sielken. 1994a. "A Distributional Approach to Characterizing Low-Dose Cancer Risk." *Risk Analysis* 14(1):25-34.
- Evans, J.S., G.M. Gray, R.L. Sielken, Jr., A.E. Smith, C. Valdez-Flores, and J.D. Graham. 1994b. "Use of Probabilistic Expert Judgment in Uncertainty Analysis of Carcinogenic Potency." *Regulatory Toxicology and Pharmacology* 20:25-36.
- Hawkins, N.C., and J.S. Evans. 1989. "Subjective Estimation of Toluene Exposures: A Calibration Study of Industrial Hygienists." *Applied. Ind. Hygiene* 4:61-68.
- Hawkins, N.C., and J.D. Graham. 1988. "Expert Scientific Judgment and Cancer Risk Assessment: A Pilot Study of Pharmacokinetic Data." *Risk Analysis* 8(4):615-625.
- Hawkins, N.C. and J.D. Graham. 1990. "Expert Scientific Judgment and Cancer Risk Assessment: A Pilot Study of Pharmacokinetic Data," Risk Analysis. 8: 615-625.

- Industrial Economics, Incorporated (IEc). March 31, 1994. Memorandum to Jim DeMocker, Office of Air and Radiation, Office of Policy Analysis and Review, U.S. Environmental Protection Agency.
- Industrial Economics, Incorporated (IEc). 2004. "An Expert Judgment Study of the Concentration-Response Relationship Between PM2.5 Exposure and Mortality," available at: <www.epa.gov/ttn/ecas/benefits.html>.
- Industrial Economics, Incorporated (IEc). April 2004. "Expert Judgment Assessment of the Relationship Between PM_{2.5} Exposure and Mortality." Available at: <a href="https://www.epa.gov/ttn/ecas/benefits.html">www.epa.gov/ttn/ecas/benefits.html</a>.
- Krewski, D., R.T. Burnett, M.S. Goldbert, K. Hoover, J. Siemiatycki, M. Jerrett, M.
   Abrahamowicz, and W.H. White. July 2000. *Reanalysis of the Harvard Six Cities* Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute, Cambridge MA.
- Krewski, D., S.N. Ras, J.M. Zielmski, and P.K. Hopke. 1999. "Characterization of Uncertainty and Variability in Residential Radon Cancer Risks." Ann. N.Y. Acad. Sci. 895:245-272.
- Krupnick, A.J., and M.L. Cropper. 1992. "The Effect of Information on Health Risk Valuations." *Journal of Risk and Uncertainty* 5(2):29-48.
- Manne, A.S., and R.G. Richels. 1994. "The Costs of Stabilizing Global CO2 Emissions: A Probabilistic Analysis Based on Expert Judgments." *The Energy Journal* 15(1):31-56.
- Mansfield, C. 2004. "Peer Review of Expert Elicitation." <www.epa.gov/ttn/ ecas/benefits.html>. Research Triangle Park, NC: RTI International.
- McCurdy, T., and H. Richmond. 1983. Description of the OAQPS Risk Program and the ongoing Lead NAAQS Risk Assessment Project. Paper 83-74.1. Presented at the 76th Annual Meeting of the Air Pollution Control Association, June 19-24, Atlanta, Georgia. As cited in NAS.
- Morgan, M.G., S.C. Morris, M. Henrion, D.A. Amaral, and W.R. Rish. 1984. "Technical Uncertainty in Quantitative Policy Analysis - A Sulfur Air Pollution Example." *Risk Analysis* 4:201-216.
- Morgan, G., and M. Henrion. 1990. Uncertainty; A Guide to Dealing with Uncertainty in Quantitative Risk and Policy Analysis. Cambridge University Press, Cambridge.

- Morgan, M.G., and D.W. Keith. 1995. "Subjective Judgments by Climate Experts." *Environmental Science and Technology* 29:468A-476A.
- National Academy of Sciences/National Research Council (NAS). 2002. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. The National Academies Press: Washington, D.C.
- Neumann, J.E., M.T. Dickie, and R.E. Unsworth. March 31, 1994. "Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis—Draft Valuation Document." Industrial Economics Incorporated (IEc) Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review.
- Nordhaus, W.D. 1994. "Expert Opinion on Climatic Change." *American Scientist* 82:45-51.
- North, W., and M.W. Merkhofer. 1976. "A Methodology for Analyzing Emission Control Strategies." *Comput. Oper. Res.* 3:187-207.
- Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- Reilly, J., P.H. Stone, C.E. Forest, M.D. Webster, H.E. Jacoby, and R.G. Prinn. 2001. "Climate Change. Uncertainty and Climate Change Assessments." *Science* 293(5529):430-433.
- Rowe, R.D., and L.G. Chestnut. 1986. "Oxidants and Asthmatics in Los Angeles: A Benefits Analysis—Executive Summary." Prepared by Energy and Resource Consultants, Inc. Report to the U.S. Environmental Protection Agency, Office of Policy Analysis. EPA-230-09-86-018. Washington, DC.
- Russell, M.W., D.M. Huse, S. Drowns, E.C. Hamel, and S.C. Hartz. 1998. "Direct Medical Costs of Coronary Artery Disease in the United States." *American Journal of Cardiology* 81(9):1110-1115.
- Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista, and W.B. Saunders. 1997. "A National Estimate of the Economic Costs of Asthma." *American Journal* of Respiratory and Critical Care Medicine 156(3 Pt 1):787-793.

- Stanford, R., T. McLaughlin, and L.J. Okamoto. 1999. "The Cost of Asthma in the Emergency Department and Hospital." *American Journal of Respiratory and Critical Care Medicine* 160(1):211-215.
- Tolley, G.S. et al. January 1986. Valuation of Reductions in Human Health Symptoms and Risks. University of Chicago. Final Report for the U.S. Environmental Protection Agency.
- U.S. Environmental Protection Agency (EPA). 1999. The Benefits and Costs of the Clean Air Act, 1990-2010. Prepared for U.S. Congress by U.S. EPA, Office of Air and Radiation/Office of Policy Analysis and Review, Washington, DC, November; EPA report no. EPA-410-R-99-001.
- U.S. Environmental Protection Agency (EPA). September 2000. *Guidelines for Preparing Economic Analyses*. EPA 240-R-00-003.
- U.S. Office of Management and Budget (OMB). October 1992. "Guidelines and Discount Rates for Benefit-Cost Analysis of Federal Programs." Circular No. A-94.
- U.S. Nuclear Regulatory Commission (NRC). 1996. "Branch Technical Position on the Use of Expert Elicitation in the High-Level Radioactive Waste Program." November, 1996.
- Viscusi, V.K., and J.E. Aldy. 2003. "The Value of a Statistical Life: A Critical Review of Market Estimates Throughout the World." *Journal of Risk and Uncertainty* 27(1):5-76.
- Viscusi, W.K., W.A. Magat, and J. Huber. 1991. "Pricing Environmental Health Risks: Survey Assessments of Risk-Risk and Risk-Dollar Trade-Offs for Chronic Bronchitis." *Journal of Environmental Economics and Management* 21:32-51.
- Walker, K.D., J.S. Evans, and D. MacIntosh. 2001. "Use of Expert Judgment in Exposure Assessment. Part 1. Characterization of Personal Exposure to Benzene." *J Expo Anal Environ Epidemiol* 11(4):308-22.
- Walker, K.D., P. Catalano, J.K. Hammitt, and J.S. Evans. 2003. "Use of Expert Judgment in Exposure Assessment: Part 2. Calibration of Expert Judgments about Personal Exposures to Benzene." *J Expo Anal Environ Epidemiol* 13(1):1-16.
- Whitfield, R.G., and T.S. Wallsten. 1989. "A Risk Assessment for Selected Lead-induced Health Effects: An Example of a General Methodology." *Risk Analysis* 9(2):197-207.

- Whitfield, R.G., T.S. Wallsten, R. L. Winkler, H.M. Richmond, and S.R. Hayes. 1991.
   Assessing the Risks of Chronic Lung Injury Attributable to Long-Term Ozone
   Exposure. Argonne National Laboratory Report ANL/EAIS-2. NTIS/DE91016814.
   Argonne, IL. July.
- Winkler, R.L., T.S. Wallsten, R.G. Whitfield, H.M. Richmond, S.R. Hayes, and A.S. Rosenbaum. 1995. "An Assessment of the Risk of Chronic Lung Injury Attributable to Long-term Ozone Exposure." *Operations Research* 43(1):19-28.
- Wittels, E.H., J.W. Hay, and A.M. Gotto, Jr. 1990. "Medical Costs of Coronary Artery Disease in the United States." *American Journal of Cardiology* 65(7):432-440.

Wright, G., and P. Ayton (eds.). 1994. Subjective Probability. John Wiley, Chichester.

#### **APPENDIX C**

## SENSITIVITY ANALYSES OF THE KEY PARAMETERS IN THE BENEFITS ANALYSIS

The primary analysis presented in Chapter 4 is based on our current interpretation of the scientific and economic literature. That interpretation requires judgments regarding the best available data, models, and modeling methodologies and the assumptions that are most appropriate to adopt in the face of important uncertainties. The majority of the analytical assumptions used to develop the primary estimates of benefits have been reviewed and approved by EPA's SAB. Both EPA and the SAB recognize that data and modeling limitations as well as simplifying assumptions can introduce significant uncertainty into the benefit results and that alternative choices exist for some inputs to the analysis, such as the mortality C-R functions.

This appendix supplements our primary estimates of benefits with a series of sensitivity calculations that use other sources of health effect estimates and valuation data for key benefits categories. These supplemental estimates examine sensitivity to both valuation issues (e.g., the appropriate income elasticity) and for physical effects issues (e.g., possible recovery from chronic illnesses). These supplemental estimates are not meant to be comprehensive. Rather, they reflect some of the key issues identified by EPA or commentors as likely to have a significant impact on total benefits. The individual adjustments in the tables should not simply be added together because 1) there may be overlap among the alternative assumptions and 2) the joint probability among certain sets of alternative assumptions may be low.

#### C.1 Premature Mortality—Long-Term Exposure

Reduction in the risk of premature mortality is the most important PM-related health outcome in terms of contribution to dollar benefits in the analysis for this rule. There are at least three important analytical assumptions that may significantly impact the estimates of the number and valuation of avoided premature mortalities. These include selection of the C-R function, structure of the lag between reduced exposure and reduced mortality risk, and effect thresholds. Results of this set of sensitivity analyses are presented in Table C-1.

 Table C-1. Sensitivity of Benefits of Premature Mortality Reductions to Alternative

 Assumptions (Relative to Primary Estimate Benefits of the Final CAIR)

		Avoided Incidences		Value (mil	lion 1999\$) ^b
D	escription of Sensitivity Analysis	2010	2015	2010	2015
Alternative C	oncentration-Response Functions for PM-Related	l Premature Mo	ortality		
Pope/ACS St					
Lur	ng Cancer	2,000	2,700	\$11,000	\$14,000
Cardiopulmonary		9,700	13,000	\$51,000	\$67,000
Krewski/Harvard Six-Cities Study		29,000	38,000	\$150,000	\$200,000
Alternative L	ag Structures for PM-Related Premature Mortalit	у			
None	Incidences all occur in the first year	13,000	17,000	\$74,000	\$102,000
8-year	Incidences all occur in the 8 th year				
	3% discount rate	13,000	17,000	\$60,000	\$83,000
	7% discount rate	13,000	17,000	\$46,000	\$64,000
15-year	Incidences all occur in the 15 th year				
	3% discount rate	13,000	17,000	\$49,000	\$68,000
	7% discount rate	13,000	17,000	\$29,000	\$40,000
Alternative Segmented	20 percent of incidences occur in 1 st year, 50 percent in years 2 to 5, and 30 percent in years 6 to 20				
	3% discount rate	13,000	17,000	\$65,000	\$90,000
	7% discount rate	13,000	17,000	\$52,000	\$71,000
5-Year Distributed	50 percent of incidences occur in years 1 and 2 and 50 percent in years 2 to 5				
	3% discount rate	13,000	17,000	\$71,000	\$97,000
	7% discount rate	13,000	17,000	\$66,000	\$91,000
Exponential	Incidences occur at an exponentially declining rate following year of change in exposure				
	3% discount rate	13,000	17,000	\$71,000	\$75,000
	7% discount rate	13,000	17,000	\$65,000	\$90,000
Alternative T	hresholds				
	No Threshold (base estimate)	13,000	17,000	\$67,000	\$93,000
	5 μg/m ³	13,000	17,000	\$67,000	\$93,000
	10 µg/m ³	11,000	14,000	\$59,000	\$78,000
	15 μg/m ³	780	690	\$4,100	\$3,800
	20 µg/m ³	0	0	\$0	\$0
	25 µg/m ³	0	0	\$0	\$0

^a Incidences rounded to two significant digits.

^b Dollar values rounded to two significant digits.

^c Note that the sum of lung cancer and cardiopulmonary deaths will not be equal to the total all-cause death estimate. Some residual mortality is associated with long-term exposures to  $PM_{2.5}$  that is not captured by the cardiopulmonary and lung cancer categories.

#### C.1.1 Alternative C-R Functions

Following the advice of the most recent EPA SAB-HES, we used the Pope et al. (2002) all-cause mortality model to derive our primary estimate of avoided premature mortality (EPA-SAB-COUNCIL-ADV-04-002, 2004). While the SAB-HES "recommends that the base case rely on the Pope et al. (2002) study and that EPA use total mortality concentration-response functions (C-R), rather than separate cause-specific C-R functions, to calculate total PM mortality cases," (EPA-SAB-COUNCIL-ADV-04-002, 2004, p.2) they also suggested that "the cause-specific estimates can be used to communicate the relative contribution of the main air pollution related causes of death." (EPA-SAB-COUNCIL-ADV-04-002, 2004, p.18) As such, in Table C-1 we provide the estimates of cardiopulmonary and lung cancer deaths based on the Pope et al. (2002) study.

In addition, the SAB-HES noted that the ACS cohort used in Pope et al. (2002) "has some inherent deficiencies, in particular the imprecise exposure data, and the nonrepresentative (albeit very large) population" (EPA-SAB-COUNCIL-ADV-04-002, 2004, p.18). The SAB-HES suggests that while not necessarily a better study, the ACS is a prudent choice for the primary estimate. They go on to note that "the Harvard Six-Cities C-R functions are valid estimates on a more representative, although geographically selected, population, and its updated analysis has not yet been published. The Six-Cities estimates may be used in a sensitivity analysis to demonstrate that, with different but also plausible selection criteria for C-R functions, benefits may be considerably larger than suggested by the ACS study" (EPA-SAB-COUNCIL-ADV-04-002, 2004, p.18). In previous advice, the SAB has noted that "the [Harvard Six-Cities] study had better monitoring with less measurement error than did most other studies" (EPA-SAB-COUNCIL-ADV-99-012, 1999). The demographics of the ACS study population (i.e., largely white and middle to upper middle-class) may also produce a downward bias in the estimated PM mortality coefficient, because a variety of analyses indicate that the effects of PM tend to be significantly greater among groups of lower socioeconomic status (Krewski et al., 2000), although the cause of this difference has not been identified. The Harvard Six-Cities study also covered a broader age category (25 and older compared to 30 and older in the ACS study). We emphasize that, based on our understanding of the relative merits of the two datasets, the Pope et al. (2002) ACS model based on mean  $PM_{2.5}$  levels in 63 cities is the most appropriate model for analyzing the premature mortality impacts of CAIR. Thus it is used for our primary estimate of this important health effect.

#### C.1.2 Alternative Lag Structures

Over the last ten years, there has been a continuing discussion and evolving advice regarding the timing of changes in health effects following changes in ambient air pollution. It has been hypothesized that some reductions in premature mortality from exposure to ambient PM_{2.5} will occur over short periods of time in individuals with compromised health status, but other effects are likely to occur among individuals who, at baseline, have reasonably good health that will deteriorate because of continued exposure. No animal models have yet been developed to quantify these cumulative effects, nor are there epidemiologic studies bearing on this question. The SAB-HES has recognized this lack of direct evidence. However, in early advice, they also note that "although there is substantial evidence that a portion of the mortality effect of PM is manifest within a short period of time, i.e., less than one year, it can be argued that, if no lag assumption is made, the entire mortality excess observed in the cohort studies will be analyzed as immediate effects, and this will result in an overestimate of the health benefits of improved air quality. Thus some time lag is appropriate for distributing the cumulative mortality effect of PM in the population" (EPA-SAB-COUNCIL-ADV-00-001, 1999, p. 9). In recent advice, the SAB-HES suggests that appropriate lag structures may be developed based on the distribution of cause-specific deaths within the overall all-cause estimate (EPA-SAB-COUNCIL-ADV-04-002, 2004). They suggest that diseases with longer progressions should be characterized by longer-term lag structures, while air pollution impacts occurring in populations with existing disease may be characterized by shorter-term lags.

A key question is the distribution of causes of death within the relatively broad categories analyzed in the long-term cohort studies. Although it may be reasonable to assume the cessation lag for lung cancer deaths mirrors the long latency of the disease, it is not at all clear what the appropriate lag structure should be for cardiopulmonary deaths, which include both respiratory and cardiovascular causes. Some respiratory diseases may have a long period of progression, while others, such as pneumonia, have a very short duration. In the case of cardiovascular disease, there is an important question of whether air pollution is causing the disease, which would imply a relatively long cessation lag, or whether air pollution is causing premature death in individuals with preexisting heart disease, which would imply very short cessation lags. The SAB-HES provides several recommendations for future research that could support the development of defensible lag structures, including using disease-specific lag models and constructing a segmented lag distribution to combine differential lags across causes of death (EPA-SAB-COUNCIL-ADV-04-002, 2004). The SAB-HES indicated support for using "a Weibull distribution or a

simpler distributional form made up of several segments to cover the response mechanisms outlined above, given our lack of knowledge on the specific form of the distributions" (EPA-SAB-COUNCIL-ADV-04-002, 2004, p. 24). However, they noted that "an important question to be resolved is what the relative magnitudes of these segments should be, and how many of the acute effects are assumed to be included in the cohort effect estimate" (EPA-SAB-COUNCIL-ADV-04-002, 2004, p. 24-25). Since the publication of that report in March 2004, EPA has sought additional clarification from this committee. In its followup advice provided in December 2004, this SAB suggested that until additional research has been completed, EPA should assume a segmented lag structure characterized by 30 percent of mortality reductions occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in PM_{2.5}, and 20 percent occurring evenly over the years 6 to 20 after the reduction in PM_{2.5} (EPA-COUNCIL-LTR-05-001, 2004). The distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to 5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. Furthermore, in their advisory letter, the SAB-HES recommended that EPA include sensitivity analyses on other possible lag structures. In this appendix, we investigate the sensitivity of premature mortality-reduction related benefits to alternative cessation lag structures, noting that ongoing and future research may result in changes to the lag structure used for the primary analysis.

In previous advice from the SAB-HES, they recommended an analysis of 0-, 8-, and 15-year lags, as well as variations on the proportions of mortality allocated to each segment in the segmented lag structure (EPA-SAB-COUNCIL-ADV-00-001, 1999, (EPA-COUNCIL-LTR-05-001, 2004). The 0-year lag is representative of EPA's assumption in previous RIAs. The 8- and 15-year lags are based on the study periods from the Pope et al. (1995) and Dockery et al. (1993) studies, respectively.¹ However, neither the Pope et al. nor Dockery et al. studies assumed any lag structure when estimating the relative risks from PM exposure. In fact, the Pope et al. and Dockery et al. analyses do not supporting or refute the existence of a lag. Therefore, any lag structure applied to the avoided incidences estimated from either of these studies will be an assumed structure. The 8- and 15-year lags implicitly assume that all premature mortalities occur at the end of the study periods (i.e., at 8 and 15 years).

¹Although these studies were conducted for 8 and 15 years, respectively, the choice of the duration of the study by the authors was not likely due to observations of a lag in effects but is more likely due to the expense of conducting long-term exposure studies or the amount of satisfactory data that could be collected during this time period.

In addition to the simple 8- and 15-year lags, we have added three additional sensitivity analyses examining the impact of assuming different allocations of mortality to the segmented lag of the type suggested by the SAB-HES. The first sensitivity analysis assumes that more of the mortality impact is associated with chronic lung diseases or lung cancer and less with acute cardiopulmonary causes. This illustrative lag structure is characterized by 20 percent of mortality reductions occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in  $PM_{2.5}$ , and 30 percent occurring evenly over the years 6 to 20 after the reduction in  $PM_{2.5}$ . The second sensitivity analysis assumes the 5-year distributed lag structure used in previous analyses, which is equivalent to a three-segment lag structure with 50 percent in the first 2-year segment, 50 percent in the second 3-year segment, and 0 percent in the 6- to 20-year segment. The third sensitivity analysis analysis assumes a negative exponential relationship between reduction in exposure and reduction in mortality risk. This structure is based on an analysis by Röösli et al. (2004), which estimates the percentage of total mortality impact in each period t as

% Mortality Reduction (t) = 
$$\frac{\left[(RR-1)e^{-0.5t}+1\right]-1}{\sum_{t=1}^{\infty}\left[(RR-1)e^{-0.5t}+1\right]-1}.$$
 (C.1)

The Röösli et al. (2004) analysis derives the lag structure by calculating the rate constant (-0.5) for the exponential lag structure that is consistent with both the relative risk from the cohort studies and the change in mortality observed in intervention type studies (e.g., Pope et al. [1992] and Clancy et al. [2002]). This is the only lag structure examined that is based on empirical data on the relationship between changes in exposure and changes in mortality. However, the analysis has not yet been peer reviewed and is thus not yet appropriate for adoption in the primary analysis.

The estimated impacts of alternative lag structures on the monetary benefits associated with reductions in PM-related premature mortality (estimated with the Pope et al. ACS impact function) are presented in Table C-1. These estimates are based on the value of statistical lives saved approach (i.e., \$5.5 million per incidence) and are presented for both a 3 and 7 percent discount rate over the lag period.

#### C.1.3 Thresholds

Although the consistent advice from EPA's SAB² has been to model premature mortality associated with PM exposure as a nonthreshold effect, that is, with harmful effects to exposed populations regardless of the absolute level of ambient PM concentrations. EPA's most recent  $PM_{2.5}$  Criteria Document concludes that "the available evidence does not either support or refute the existence of thresholds for the effects of PM on mortality across the range of concentrations in the studies" (U.S. EPA, 2004, p. 9-44). Some researchers have hypothesized the presence of a threshold relationship. The nature of the hypothesized relationship is that there might exist a PM concentration level below which further reductions no longer yield premature mortality reduction benefits.³

We constructed a sensitivity analysis by assigning different cutpoints below which changes in  $PM_{2.5}$  are assumed to have no impact on premature mortality. The sensitivity analysis illustrates how our estimates of the number of premature mortalities in the primary estimate might change under a range of alternative assumptions for a PM mortality threshold. If, for example, there were no benefits of reducing PM concentrations below the  $PM_{2.5}$  standard of 15 µg/m³, our estimate of the total number of avoided PM-related premature mortalities in 2015 from the primary analysis would be reduced by approximately 96 percent, from approximately 17,000 annually to approximately 700 annually. The recent NRC report stated that "for pollutants such as  $PM_{10}$  and  $PM_{2.5}$ , there is no evidence for any departure of linearity in the observed range of exposure, nor any indication of a threshold" (NRC, 2002, p. 109). At a threshold of 10 µg, approximately the 20th percentile of observed concentrations in the Pope et al. (2002) study, mortality impacts would be reduced by only 16 percent to approximately 14,000 annually.

²The advice from the 2004 SAB-HES (EPA-SAB-COUNCIL-ADV-04-002) is characterized by the following: "For the studies of long-term exposure, the HES notes that Krewski et al. (2000) have conducted the most careful work on this issue. They report that the associations between PM_{2.5} and both all-cause and cardiopulmonary mortality were near linear within the relevant ranges, with no apparent threshold. Graphical analyses of these studies (Dockery et al., 1993, Figure 3, and Krewski et al., 2000, page 162) also suggest a continuum of effects down to lower levels. Therefore, it is reasonable for EPA to assume a no threshold model down to, at least, the low end of the concentrations reported in the studies."

³The illustrative example in Appendix B presents the potential implications of assuming some probability of a threshold on the benefits estimate.

have not conducted at this time might examine the potential for a nonlinear relationship at lower exposure levels.⁴

One important assumption that we adopted for the threshold sensitivity analysis is that no adjustments are made to the shape of the C-R function above the assumed threshold. Instead, thresholds were applied by simply assuming that any changes in ambient concentrations below the assumed threshold have no impacts on the incidence of premature mortality. If there were actually a threshold, then the shape of the C-R function would likely change and there would be no health benefits to reductions in PM below the threshold. However, as noted by the NRC, "the assumption of a zero slope over a portion of the curve will force the slope in the remaining segment of the positively sloped concentration-response function to be greater than was indicated in the original study" and that "the generation of the steeper slope in the remaining portion of the concentration-response function may fully offset the effect of assuming a threshold." The NRC suggested that the treatment of thresholds should be evaluated in a formal uncertainty analysis.

#### C.1.3 Summary of Results

The results of these sensitivity analyses demonstrate that choice of effect estimate can have a large impact on benefits, potentially doubling benefits if the effect estimate is derived from the HEI reanalysis of the Harvard Six-Cities data (Krewski et al., 2000). Because of discounting of delayed benefits, the lag structure may also have a large impact on monetized benefits, reducing benefits by 30 percent if an extreme assumption that no effects occur until after 15 years is applied. However, for most reasonable distributed lag structures, differences in the specific shape of the lag function have relatively small impacts on overall benefits. For example, the overall impact of moving from the previous 5-year distributed lag to the segmented lag recommended by the SAB-HES in 2004 in the primary estimate is relatively modest, reducing benefits by approximately 5 percent when a 3 percent discount rate is used and 15 percent when a 7 percent discount rate is used. If no lag is assumed, benefits are increased by around 10 percent relative to the segmented lag with a 3 percent discount rate and 30 percent with a 7 percent discount rate. Benefits are more sensitive to assumptions regarding the potential for a threshold. The threshold sensitivity analysis indicates that over 84 percent of the premature mortality-related benefits are due to changes

⁴The pilot expert elicitation discussed in Appendix B provides some information on the impact of applying nonlinear and threshold-based C-R functions.

in  $PM_{2.5}$  concentrations occurring above 10  $\mu$ g/m³, and around 5 percent are due to changes above 15  $\mu$ g/m³, the current  $PM_{2.5}$  standard.

## C.2 Other Health Endpoint Sensitivity Analyses

#### C.2.1 Ozone-Related Mortality

To provide the reader with a fuller understanding of the health effects associated with reductions in air pollution associated with the final CAIR, this set of sensitivity estimates examines the potential impact of reductions in ozone on incidence of premature mortality. Although PM is the air pollutant most clearly associated with premature mortality, recent research suggests that short-term ozone exposure likely contributes to premature death. Although the CAIR analysis uses cohort studies to characterize the effect of PM_{2.5} on premature mortality, such cohort studies have for the most part not documented an association between ozone exposure and premature mortality. Time series studies, which look specifically at the effects of short-term exposures, have documented effects on premature mortality from both PM_{2.5} and ozone, although results have been mixed. Several recent analyses have found consistent statistical associations between short-term ozone exposure and increased mortality (Bell et al., 2004; Fairly et al., 2003; Thurston and Ito, 2001; Toulomi et al., 1997). The most recent of these, Bell et al. (2004), used the extensive National Morbidity, Mortality, and Air Pollution Study database to examine associations between ozone and premature mortality in 95 U.S. urban communities. They found that on average, short-term changes in ozone are significantly associated with premature mortality, and that the association is robust to adjustment for particulate matter, weather, and seasonality.

Although they do not constitute a database as extensive as that for PM, these recent studies provide supporting evidence for including mortality in ozone health benefits analyses. In a 2001 analysis, Thurston and Ito reviewed previously published time-series studies examining the effect of daily ozone levels on daily mortality. Thurston and Ito hypothesized that much of the variability in published estimates of the ozone/mortality effect could be explained by how well each model controlled for the influence of weather, an important confounder and that earlier studies, which used less-sophisticated approaches to controlling for weather, consistently underpredicted the ozone/mortality effect.

Thurston and Ito (2001) also found that models incorporating a nonlinear temperature specification appropriate for the "U-shaped" nature of the temperature/mortality relationship (i.e., increased deaths at both very low and very high temperatures) produced ozone/

mortality effect estimates that were both more strongly positive (an increase in relative risk over the pooled estimate for all studies evaluated, from 1.036 to 1.056) and consistently statistically significant. Further accounting for the interaction effects between temperature and relative humidity strengthened the positive effect. Including a PM index to control for PM/mortality effects had little effect on these results, suggesting a relationship between ozone and mortality independent of that for PM. However, most of the studies Thurston and Ito examined controlled only for PM₁₀ or broader measures of particles and did not directly control for PM_{2.5}. As such, there still may be potential for confounding of PM_{2.5} and ozone mortality effects, given that ozone and PM_{2.5} are highly correlated during summer months in some areas.

Two recent World Health Organization reports concluded that recent epidemiological studies have strengthened the evidence that there are short-term  $O_3$  effects on mortality and respiratory morbidity (Anderson et al. 2004; WHO, 2003). In addition, Levy et al. (2001) assessed the epidemiological evidence regarding the link between short-term exposures to ozone and premature mortality. Based on four U.S. studies (Kellsall et al., 1997; Moolgavkar et al., 1995; Ito and Thurston, 1996; Moolgavkar, 2000), they concluded that an appropriate pooled effect estimate is a 0.5 percent increase in premature deaths per 10  $\mu$ g/m³ increase in 24-hour average ozone concentrations, with a 95 percent confidence interval between 0.3 percent and 0.7 percent.

In its September 2001 advisory on the draft analytical blueprint for the second Section 812 prospective analysis, the SAB HES cited the Thurston and Ito study as a significant advance in understanding the effects of ozone on daily mortality and recommended re-evaluation of the ozone mortality endpoint for inclusion in the next prospective study (EPA-SAB-COUNCIL-ADV-01-004, 2001). Based on these new analyses and recommendations, EPA sponsored three independent meta-analyses of the ozone-mortality epidemiology literature to inform a determination on including this important health endpoint. Publication of these meta-analyses will significantly enhance the scientific defensibility of benefits estimates for ozone, which include the benefits of premature mortality reductions.

We estimate ozone mortality for this sensitivity analysis with the recognition that the exact magnitude of the effects estimate is subject to continuing uncertainty. As we have done in the sensitivity analyses for prior RIAs, such as for the non-road diesel rule, we used results from three U.S. studies to calculate the base-case ozone mortality estimate (Table C-2). We selected these studies (Ito and Thurston, 1996; Moolgavkar et al., 1995;

	Avoided Incidences		Monetized Value (Million 1999\$)	
Description of Sensitivity Analysis	2010	2015	2010	2015
Mortality from short-term ozone exposure ^b				
Ito and Thurston (1996)	290	850	\$1,700	\$5,200
Moolgavkar et al. (1995)	150	410	\$900	\$2,500
Samet et al. (1997)	230	630	\$1,400	\$3,800
Pooled estimate (random effects weights)	190	520	\$1,100	\$3,200

#### Table C-2. Sensitivity Estimates for Ozone-Related Premature Mortality

^a All estimates rounded to two significant digits.

^b Mortality valued using base estimate of \$5.5 million per premature statistical death, adjusted for income growth.

Samet et al., 1997) based on the logic that the demographic and environmental conditions existing when these studies were conducted would, on average, be most similar (relative to international studies) to the conditions prevailing when the CAIR would be implemented. We excluded a fourth U.S. study by Kinney et al. (1995) because, as Levy et al. (2001) noted, that study included only a linear term for temperature. Because Kinney et al. (1995) found no significant ozone effect, including this study would lead to an underestimate of true mortality impacts and increase the uncertainty surrounding the estimated mortality reductions.

We then estimated the change in mortality incidence resulting from applying the effect estimate from each study and combined the results using a random-effects weighting procedure that accounts for both the precision of the individual effect estimates and betweenstudy variability (see Appendix D for more details on this method for combining results). However, it is important to note that this procedure only captures the uncertainty in the underlying epidemiological work and does not capture other sources of uncertainty, such as that in estimating air pollution exposure (Levy et al., 2001).

Table C-2 shows that if it is assumed that ozone independently affects premature mortality, then in 2010, an additional 190 premature deaths might be avoided from reductions in ozone concentrations due to CAIR, while in 2015, an additional 520 deaths might be avoided. This would add an additional \$1.1 billion in monetized benefits in 2010, and an additional \$3.2 billion in monetized benefits in 2015.

#### C.2.2 Alternative and Supplementary Estimates

We also examined how the value for individual endpoints or total benefits would change if we were to make a different assumption about specific elements of the benefits analysis. Specifically, in Table C-3, we show the impact of alternative assumptions about other parameters, including treatment of reversals in CB, alternative impact functions for PM hospital and emergency room admissions, valuation of residential visibility, valuation of recreational visibility at Class I areas outside of the study regions examined in the Chestnut and Rowe (1990a, 1990b) study, and valuation of household soiling damages.

	Alternative			rimary Benefit nillion 2000\$)
	Calculation	Description of Estimate	2010	2015
1	Treatment of reversals in CB	Instead of omitting cases of CB that reverse after a period of time, they are treated as being cases with the lowest severity rating. The number of avoided chronic CB in 2010 increases from 6,900 to 13,000 (88%). The increase in 2015 is from 8,700 to 16,000 (84%).	+\$880	+\$1,100
2	Value of visibility changes in all Class I areas	Values of visibility changes at Class I areas in California, the Southwest, and the Southeast are transferred to visibility changes in Class I areas in other regions of the country.	+\$200	+\$200
3	Household soiling damage	Value of decreases in expenditures on cleaning are estimated using values derived from Manuel et al. (1983).	+\$280	+\$340

#### Table C-3. Additional Parameter Sensitivity Analyses

An important assumption related to chronic conditions is the possible reversal in CB incidences (row 1 of Table C-3). Reversals are defined as those cases where an individual reported having CB at the beginning of the study period but reported not having CB in follow-up interviews at a later point in the study period. Because chronic diseases are long-lasting or permanent by definition, if the disease abates in a shorter period of time it is not chronic. However, we have not captured the benefits of reducing incidences of bronchitis that are somewhere in between acute and chronic. Since chronic bronchitis may be assigned a range of severities, one way to address this is to treat reversals as cases of CB that are at the

lowest severity level. These reversals of CB thus are assigned the lowest value for CB in this sensitivity analysis, rather than omitting reversals as is the case in the primary analysis.

The alternative calculation for recreational visibility (row 2 of Table C-3) is an estimate of the full value of visibility in the entire region affected by the CAIR emission reductions. The Chestnut and Rowe (1990a) study from which the primary valuation estimates are derived only examined WTP for visibility changes in the southeastern portion of the affected region. To obtain estimates of WTP for visibility changes in the northeastern and central portion of the affected region, we have to transfer the southeastern WTP values. This introduces additional uncertainty into the estimates. However, we have taken steps to adjust the WTP values to account for the possibility that a visibility improvement in parks in one region is not necessarily the same environmental quality good as the same visibility improvement at parks in a different region. This may be due to differences in the scenic vistas at different parks, uniqueness of the parks, or other factors, such as public familiarity with the park resource. To take this potential difference into account, we adjusted the WTP being transferred by the ratio of visitor days in the two regions.

The alternative calculation for household soiling (row 5 of Table C-3) is based on the Manuel et al. (1983) study of consumer expenditures on cleaning and household maintenance. This study has been cited as being "the only study that measures welfare benefits in a manner consistent with economic principals" (Desvousges et al., 1998). However, the data used to estimate household soiling damages in the Manuel et al. study are from a 1972 consumer expenditure survey and as such may not accurately represent consumer preferences in 2015. EPA recognizes this limitation, but believes the Manuel et al. estimates are still useful in providing an estimate of the likely magnitude of the benefits of reduced PM household soiling.

#### C.3 Income Elasticity of Willingness to Pay

As discussed in Chapter 4, our estimates of monetized benefits account for growth in real GDP per capita by adjusting the WTP for individual endpoints based on the central estimate of the adjustment factor for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility). We examined how sensitive the estimate of total benefits is to alternative estimates of the income elasticities. Table C-4 lists the ranges of elasticity values used to calculate the income adjustment factors, while Table C-5 lists the ranges of corresponding adjustment factors. The results of this sensitivity analysis, giving the monetized benefit subtotals for the four benefit categories, are presented in Table C-6.

# Table C-4. Ranges of Elasticity Values Used to Account for Projected Real Income Growth^a

Benefit Category	Lower Sensitivity Bound	Upper Sensitivity Bound
Minor Health Effect	0.04	0.30
Severe and Chronic Health Effects	0.25	0.60
Premature Mortality	0.08	1.00
Visibility ^b	_	

^a Derivation of these ranges can be found in Kleckner and Neumann (1999) and Chestnut (1997). COI estimates are assigned an adjustment factor of 1.0.

^b No range was applied for visibility because no ranges were available in the current published literature.

# Table C-5. Ranges of Adjustment Factors Used to Account for Projected Real Income Growth^a

	Lower Sensitivity Bound		Upper Sensitivity Bound		
Benefit Category	2010	2015	2010	2015	
Minor Health Effect	1.010	1.015	1.074	1.114	
Severe and Chronic Health Effects	1.061	1.094	1.153	1.241	
Premature Mortality	1.019	1.029	1.269	1.437	
Visibility ^b					

^a Based on elasticity values reported in Table C-4, U.S. Census population projections, and projections of real GDP per capita.

^b No range was applied for visibility because no ranges were available in the current published literature.

	Benefits in Millions of 1999\$						
	Lower Sens	itivity Bound	Upper Sensi	itivity Bound			
<b>Benefit</b> Category	2010	2015	2010	2015			
Minor Health Effect	\$640	\$830	\$670	\$880			
Severe and Chronic Health Effects	\$3,900	\$5,100	\$4,200	\$5,600			
Premature Mortality	\$63,000	\$83,000	\$78,000	\$120,000			
Visibility and Other Welfare Effects ^b	\$1,100	\$1,800	\$1,100	\$1,800			
Total Benefits	\$68,000	\$91,000	\$84,000	\$120,000			

#### Table C-6. Sensitivity Analysis of Alternative Income Elasticities^a

^a All estimates rounded to two significant digits.

^b No range was applied for visibility because no ranges were available in the current published literature.

Consistent with the impact of mortality on total benefits, the adjustment factor for mortality has the largest impact on total benefits. The value of mortality in 2015 ranges from 90 percent to 130 percent of the primary estimate based on the lower and upper sensitivity bounds on the income adjustment factor. The effect on the value of minor and chronic health effects is much less pronounced, ranging from 98 percent to 105 percent of the primary estimate for minor effects and from 93 percent to 106 percent for chronic effects.

#### C.4 References

- Anderson, H.R., R.W. Atkinson, J.L. Peacock, L. Marston, and K. Konstantinou. 2004.
   Meta-analysis of Time-series Studies and Panel Studies of Particulate Matter (PM) and Ozone (O₃): Report of a WHO Task Group. Copenhagen: World Health Organization.
- Bell, M.L., A. McDermott, S.L. Zeger, J.M. Samet, and F. Dominici. 2004. "Ozone and Short-term Mortality in 95 U.S. Urban Communities, 1987-2000." *Journal of the American Medical Association* 292:2372-2378.
- Chestnut, L.G. 1997. "Draft Memorandum: Methodology for Estimating Values for Changes in Visibility at National Parks." April 15.

- Chestnut, L.G., and R.D. Rowe. 1990a. Preservation Values for Visibility Protection at the National Parks: Draft Final Report. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC and Air Quality Management Division, National Park Service, Denver, CO.
- Chestnut, L.G., and R.D. Rowe. 1990b. "A New National Park Visibility Value Estimates." In Visibility and Fine Particles, Transactions of an AWMA/EPA International Specialty Conference, C.V. Mathai, ed. Air and Waste Management Association, Pittsburgh.
- Clancy, L., P. Goodman, H. Sinclair, and D.W. Dockery. 2002. "Effect of Air-pollution Control on Death Rates in Dublin, Ireland: An Intervention Study." *Lancet* Oct 19;360(9341):1210-4.
- Desvousges, W.H., F.R. Johnson, and H.S. Banzhaf. 1998. Environmental Policy Analysis With Limited Information: Principles and Applications of the Transfer Method (New Horizons in Environmental Economics.) Edward Elgar Pub: London.
- Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris, and F.E. Speizer. 1993. "An Association between Air Pollution and Mortality in Six U.S. Cities." *New England Journal of Medicine* 329(24):1753-1759.
- EPA-SAB-COUNCIL-ADV-00-001. October 1999. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects. Part 2.
- EPA-SAB-COUNCIL-ADV-99-012. July 1999. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects. Part 1.
- EPA-SAB-COUNCIL-ADV-01-004. September 2001. Review of the Draft Analytical Plan for EPA's Second Prospective Analysis—Benefits and Costs of the Clean Air Act 1990-2020: An Advisory by a Special Panel of the Advisory Council on Clean Air Compliance Analysis.

**US EPA ARCHIVE DOCUMENT** 

- EPA-SAB-COUNCIL-ADV-04-002. March 2004. Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis—Benefits and Costs of the Clean Air Act, 1990-2020: Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis.
- Fairley, D. 2003. "Mortality and Air Pollution for Santa Clara County, California, 1989-1996." In: *Revised Analysis of Time Series Studies of Air Pollution and Health*. Special Report. Boston: Health Effects Institute, 97-106.
- Ito, K., and G.D. Thurston. 1996. "Daily PM₁₀/Mortality Associations: An Investigations of At-Risk Subpopulations." *Journal of Exposure Analysis and Environmental Epidemiology* 6(1):79-95.
- Kellsall, J., J.M. Samet, and S.L. Zeger. 1997. "Air Pollution and Mortality in Philadelphia, 1974-1988." *American Journal of Epidemiology* 146:750-762.
- Kinney, P.L., K. Ito, and G.D. Thurston. 1995. "A Sensitivity Analysis of Mortality PM-10 Associations in Los Angeles." *Inhalation Toxicology* 7(1):59-69.
- Kleckner, N., and J. Neumann. June 3, 1999. "Recommended Approach to Adjusting WTP Estimates to Reflect Changes in Real Income." Memorandum to Jim Democker, US EPA/OPAR.
- Krewski, D., R.T. Burnett, M.S. Goldbert, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz, and W.H. White. July 2000. *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality*. Special Report to the Health Effects Institute, Cambridge MA.
- Levy, J.L., Carrothers, T.J., J.T. Tuomisto, J.K. Hammitt, and J.S. Evans. 2001. "Assessing the Public Health Benefits of Reduced Ozone Concentrations." *Environmental Health Perspectives* 109:1215-1226.
- Manuel, E.H., R.L. Horst, K.M. Brennan, W.N. Lanen, M.C. Duff, and J.K. Tapiero. 1983. Benefits Analysis of Alternative Secondary National Ambient Air Quality Standards for Sulfur Dioxide and Total Suspended Particulates, Volumes I-IV. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Research Triangle Park, NC.
- Moolgavkar, S.H., E.G. Luebeck, T.A. Hall, and E.L. Anderson. 1995. "Air Pollution and Daily Mortality in Philadelphia." *Epidemiology* 6(5):476-484.

- Moolgavkar, S.H. 2000. "Air Pollution and Hospital Admissions for Diseases of the Circulatory System in Three U.S. Metropolitan Areas." *Journal of the Air and Waste Management Association* 50:1199-1206.
- National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. The National Academies Press: Washington, D.C.
- Pope, C.A. III, J. Schwartz, and M.R. Ransom. 1992. "Daily Mortality and PM₁₀ Pollution in Utah Valley." *Arch Environ Health* 47(3):211-217.
- Pope, C.A., III, M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer, and C.W. Heath, Jr. 1995. "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults." *American Journal of Respiratory Critical Care Medicine* 151:669-674.
- Pope, C.A., III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- Röösli, M., N. Künzli, and C. Braun-Fahrländer. August 1-4, 2004. "Use of Air Pollution 'Intervention-Type' Studies in Health Risk Assessment." 16th Conference of the International Society for Environmental Epidemiology, New York.
- Samet, J.M., S.L. Zeger, J.E. Kelsall, J. Xu, and L.S. Kalkstein. March 1997. Air Pollution, Weather, and Mortality in Philadelphia 1973-1988. Cambridge, MA: Health Effects Institute.
- Thurston, G.D., and K. Ito. 2001. "Epidemiological Studies of Acute Ozone Exposures and Mortality." *J Expo Anal Environ Epidemiol*. 11(4):286-94.
- Toulomi, G., K. Katsouyanni, D. Zmirou, and J. Schwartz. 1997. "Short-term Effects of Ambient Oxidant Exposure on Mortality: A Combined Analysis within the APHEA Project." American Journal of Epidemiology 146:177-183.
- U.S. Environmental Protection Agency (EPA). 2004. Air Quality Criteria for Particulate Matter, Volume II. Office of Research and Development. EPA/600/P-99/002bF, October 2004.

 World Health Organization (WHO). 2003. Health Aspects of Air Pollution with Particulate Matter, Ozone, and Nitrogen Dioxide: Report on a WHO Working Group.
 EUR/03/5042688. Bonn, Germany: World Health Organization.

#### **APPENDIX D**

## SENSITIVITY ANALYSES OF KEY PARAMETERS IN THE COST AND ECONOMIC IMPACT ANALYSIS AND A LISTING OF IPM RUNS IN SUPPORT OF CAIR

This appendix presents results of sensitivity analyses using the IPM with alternative assumptions for the price of natural gas and the growth rate of electricity demand. In addition, a list of the IPM runs that were used in the various analyses done in support of the final CAIR is provided. Model output from each of the IPM runs listed in this memo is available in the CAIR docket and also on EPA's Web site at www.epa.gov/airmarkets/ epa-ipm.

EPA uses IPM to estimate costs and, more broadly, analyze the projected impact of air emission control policies on the electric power sector in the 48 contiguous states and the District of Columbia. IPM is a multiregional, dynamic, deterministic linear programming model of the U.S. electric power sector. IPM documentation is available in the CAIR docket and also on EPA's Web site at www.epa.gov/airmarkets/epa-ipm/.

Modeling applications of IPM produce forecasts for model plants (i.e., clusters of real-life EGUs with similar characteristics). The model plant projections can be used to produce parsed results, which are unit-level results derived from the model plant projections. Projections for individual plants are based on data currently available and modeling parameters that are simplifications of the real world. It is likely that some future actions regarding individual plants could differ from model projections of actions. However, the aggregate impacts are expected to be appropriately characterized by the model. Where appropriate, EPA produced parsed results from IPM runs for use in analyzing the air quality impacts of CAIR.

#### D.1 Effects of Change in Assumptions for Natural Gas Prices and Electricity Growth

Sensitivity analyses were performed using projections from the 2004 Annual Energy Outlook produced by the Energy Information Administration (EIA). EPA used EIA estimates for the difference between natural gas prices and coal prices, which we have shorthanded as "EIA natural gas prices," as well as EIA's projection of electricity growth. These particular assumptions involve considering the higher differential between minemouth coal and wellhead natural gas prices. For the years 2010, 2015, and 2020, there was a higher differential of \$0.25 mmBtu, \$0.42 mmBtu, and \$0.38 mmBtu, respectively. The electricity growth was changed to match EIA's growth of 1.8 percent a year rather than EPA's growth of 1.6 percent.

Total annual regional costs of CAIR with EIA assumptions are in Table D-1. The costs of CAIR with EIA assumptions for natural gas prices and electricity growth in 2010 and 2015 are only slightly different from costs of CAIR without those assumptions and can be attributed to the building of new and cleaner coal-fired capacity that leads to lower overall costs (see Tables D-1 and D-2). As demand continues to grow, coal-fired generation continues to increase and requires the use of additional scrubbers. Although more pollution controls are installed using EIA assumptions, dispatch changes lead to the use of more efficient generation. The power sector is less inclined to use gas as a compliance option in the region because of the higher operating cost. Once the power sector passes the point where there is no longer excess gas capacity in the marketplace (as currently exists), new coal-fired capacity is the logical choice to meet demand. This new capacity would be built inside and outside the CAIR region.

The annualized regional cost of CAIR, as presented in Table D-1, is EPA's best assessment of the cost of implementing CAIR, assuming that States adopt the model cap and trade program. These costs are generated from rigorous economic modeling of changes in the power sector due to CAIR. This type of analysis using IPM has undergone peer review and been upheld in Federal courts. The direct cost includes, but is not limited to, capital investments in pollution controls, operating expenses of the pollution controls, investments in new generating sources, and additional fuel expenditures. EPA believes that these costs reflect, as closely as possible, the additional costs of CAIR to industry. The relatively small cost associated with monitoring emissions for affected sources is not included in the annualized cost, but EPA has done a separate analysis and estimated the cost to be less than \$42 million (see Section X. B. Paperwork Reduction Act). However, there may exist certain costs that EPA has not quantified in these estimates. These costs may include costs of transitioning to CAIR, such as the costs associated with the retirement of smaller or less efficient electricity generating units, employment shifts as workers are retrained at the same company or re-employed elsewhere in the economy, and certain relatively small permitting costs associated with Title IV that new program entrants face. Although EPA has not quantified these costs, the Agency believes that they are small compared to the quantified costs of the program on the power sector. The annualized cost estimates are the best and

Table D-1. Annual Regional Costs of CAIR with EPA and EIA Assumptions for
Natural Gas Prices and Electric Growth (Billion \$1999)

Year	<b>EPA</b> Assumptions	<b>EIA Assumptions</b>
2010	\$2.4	\$2.6
2015	\$3.6	\$3.4
2020	\$4.4	\$4.1

Source: Integrated Planning Model run by EPA.

 Table D-2. Incremental Pollution Controls under CAIR with EPA and EIA

 Assumptions for Natural Gas and Electricity Growth (Incremental GWs)

	EPA Assumptions			EIA Assumptions		
Technology	2010	2015	2020	2010	2015	2020
FGD	37	64	82	45	69	92
SCR	14	34	33	18	39	40

Source: Integrated Planning Model run by EPA.

most accurate based upon available information. At the macroeconomic level, the indirect costs and impacts of higher electricity prices on the entire economy are presented in Appendix E of the Regulatory Impact Analysis.

The marginal cost to remove additional tons of  $SO_2$  and  $NO_x$  increases slightly with EIA assumptions for natural gas and electricity growth, as more controls are installed on coal-fired units that are slightly more expensive to control. Table D-3 compares the result of sensitivity analysis to the CAIR case with EPA assumptions.

Table D-4 shows nationwide emissions of  $SO_2$  and  $NO_x$  using EIA assumptions. Coal-fired generation under CAIR increases using EIA assumptions for natural gas prices and electricity growth. Table D-5 shows the generation mix with EIA assumptions.

		2010	2015	2020
	EPA Assumptions	\$700	\$1,000	\$1,400
SO ₂	EIA Assumptions	\$800	\$1,200	\$1,500
NOx	EPA Assumptions	\$1,300	\$1,600	\$1,600
NOX	EIA Assumptions	\$1,400	\$1,700	\$1,700

Table D-3. Marginal Cost of SO₂ and NO_x Reductions under CAIR with EPA and EIA Assumptions for Natural Gas Prices and Electric Growth (\$/ton, in \$1999)

Source: Integrated Planning Model run by EPA.

Table D-4. Projected Nationwide Emissions of SO ₂ and NO _x under CAIR with EPA
and EIA Assumptions for Natural Gas and Electric Growth (Million Tons)

		SO ₂			NO _x	
	2010	2015	2020	2010	2015	2020
Base Case with EPA Assumptions	9.7	8.9	8.6	3.6	3.7	3.7
CAIR with EPA Assumptions	6.1	4.9	4.2	2.4	2.1	2.1
Base Case with EIA Assumptions	9.7	8.8	8.6	3.7	3.7	3.8
CAIR with EIA Assumptions	6.1	5.0	4.0	2.4	2.1	2.2

Source: Integrated Planning Model run by EPA.

		EP	A Assumpt	ions	<b>EIA Assumptions</b>					
	Fuel	2010	2015	2020	2010	2015	2020			
	Coal	2,198	2,242	2,410	2,243	2,638	3,048			
Base	Oil/Natural Gas	777	1,026	1,221	902	867	873			
Case	Other	1,223	1,235	1,218	1,224	1,235	1,224			
	Total	4,198	4,503	4,850	4,369	4,739	5,145			
	Coal	2,163	2,195	2,381	2,228	2,632	3,045			
	Oil/Natural Gas	809	1,072	1,250	916	871	874			
CAIR	Other	1,218	1,233	1,217	1,223	1,234	1,221			
	Total	4,190	4,499	4,847	4,367	4,738	5,141			

 Table D-5. Generation Mix under CAIR with EPA and EIA Assumptions for Natural Gas and Electric Growth (Thousand GWhs)

Note: Numbers may not add due to rounding.

Source: Integrated Planning Model run by EPA.

Coal production patterns change slightly and production for all three major coalproducing regions is higher, because coal-fired generation is a cheaper source of electricity than natural gas in most parts of the country with the higher EIA prices, even as more pollution controls are added to coal-fired generation and used to meet the additional electricity demand (see Table D-6).

Electricity prices are not greatly altered with EIA assumptions for natural gas and electricity growth (see Tables D-7 and D-8). For the CAIR region, average electricity prices are projected to be lower than current levels (2000) using both EPA and EIA assumptions for natural gas and electricity growth.

The IPM sensitivities listed in this appendix (Table D-9) that are not discussed in this document were also used in support of CAIR, and relevant discussion can be found in the appropriate section of the CAIR preamble.

 Table D-6. Coal Production for the Electric Power Sector under CAIR with EPA and EIA Assumptions for Natural Gas and Electricity Growth (Million Tons)

	Supply			EPA	Assump	tions	EIA	Assumpt	tions
	Area	2000	2003	2010	2015	2020	2010	2015	2020
	Appalachia	299	275	325	315	301	328	341	340
Base	Interior	131	135	161	162	173	161	182	247
Case	West	475	526	603	631	714	626	748	840
	National	905	936	1,089	1,109	1,188	1,115	1,271	1,428
	Appalachia	299	275	306	310	331	320	367	390
	Interior	131	135	164	193	219	174	207	260
CAIR	West	475	526	607	579	607	614	676	765
	National	905	936	1,077	1,082	1,156	1,109	1,250	1,415

Source: 2000 and 2003 data are from EIA. All projections are from the Integrated Planning Model run by EPA.

Table D-7. Retail Electricity Prices by NERC Region with the Base Case (No Further Controls) and with CAIR Using EPA Assumptions for Natural Gas and Electricity Growth (Mills/kWh)

Power			]	Base Ca	se		CAIR		Per	cent Ch	ange
Region	Primary States Included	2000	2010	2015	2020	2010	2015	2020	2010	2015	2020
ECAR	OH, MI, IN, KY, WV, PA	57.4	51.7	55.2	56.1	53.8	58.5	58.0	4.0%	5.9%	3.4%
ERCOT	TX	65.1	57.9	64.4	62.6	59.3	64.6	63.3	2.5%	0.2%	1.2%
MAAC	PA, NJ, MD, DC, DE	80.4	59.3	69.4	72.2	61.2	71.7	72.8	3.2%	3.4%	0.8%
MAIN	IL, MO, WI	61.2	52.6	57.8	61.0	54.0	60.3	62.0	2.6%	4.3%	1.7%
MAPP	MN, IA, SD, ND, NE	57.4	52.8	49.3	47.6	52.9	49.6	48.0	0.2%	0.7%	0.8%
NY	NY	104.3	82.8	87.9	88.1	83.3	88.8	88.4	0.5%	1.0%	0.3%
NE	VT, NH, ME, MA, CT, RI	89.9	77.4	83.9	82.8	77.5	84.7	83.0	0.1%	1.0%	0.2%
FRCC	FL	67.9	71.2	71.3	69.5	71.7	72.3	70.5	0.8%	1.3%	1.5%
STV	VA, NC, SC, GA, AL, MS, TN, AR, LA	59.3	56.2	55.1	55.3	57.0	56.2	56.6	1.5%	2.1%	2.3%
SPP	KS, OK, MO	59.3	54.2	57.0	56.7	54.6	57.5	57.0	0.7%	0.9%	0.6%
Regionwid	le	66.0	58.0	60.8	61.0	59.2	62.4	62.1	2.0%	2.7%	1.8%

Source: Retail Electricity Price Model run by EPA. 2000 prices are from EIA's AEO 2003.

# Table D-8. Retail Electricity Prices by NERC Region with the Base Case (No Further Controls) and with CAIR Using EIA Assumptions for Natural Gas and Electricity Growth (Mills/kWh)

Power			Base Case			CAIR			Percent Change		
Region	Primary States Included	2000	2010	2015	2020	2010	2015	2020	2010	2015	2020
ECAR	OH, MI, IN, KY, WV, PA	57.4	53.5	59.8	57.1	55.3	61.5	58.8	3.5%	3.0%	3.0%
ERCOT	TX	65.1	63.3	66.0	64.4	63.6	66.6	65.0	0.5%	0.8%	0.9%
MAAC	PA, NJ, MD, DC, DE	80.4	63.1	74.7	72.8	64.0	75.4	73.7	1.4%	1.0%	1.3%
MAIN	IL, MO, WI	61.2	54.9	63.8	62.4	55.9	65.2	63.3	1.7%	2.2%	1.5%
MAPP	MN, IA, SD, ND, NE	57.4	52.9	49.6	48.1	53.1	49.9	48.6	0.4%	0.6%	0.9%
NY	NY	104.3	89.0	91.3	87.8	89.1	91.9	88.8	0.1%	0.6%	1.1%
NE	VT, NH, ME, MA, CT, RI	89.9	85.1	85.5	81.2	84.7	85.9	81.8	-0.4%	0.5%	0.9%
FRCC	FL	67.9	72.5	74.6	73.7	73.3	75.3	74.3	1.1%	0.9%	0.9%
STV	VA, NC, SC, GA, AL, MS, TN, AR, LA	59.3	57.1	57.1	57.1	57.8	58.3	58.6	1.1%	2.0%	2.6%
SPP	KS, OK, MO	59.3	56.2	59.5	57.9	56.7	59.7	58.1	0.9%	0.3%	0.3%
Regionwid	e	66.0	60.5	64.0	62.5	61.3	65.1	63.6	1.4%	1.6%	1.8%

Source: Retail Electricity Price Model run by EPA. 2000 prices are from EIA's AEO 2003.

# Table D-9. Listing of Runs from the Integrated Planning Model Used in Analyses Done in Support of the CAIR Final Rule Analyses

Run Name	Run Description
Base Case 2004	Base case model run, which includes the national Title IV $SO_2$ cap-and-trade program; $NO_x$ SIP Call regional ozone season cap-and-trade program; and state-specific programs in Connecticut, Illinois, Maine, Massachusetts, Minnesota, Missouri, New Hampshire, New York, North Carolina, Oregon, Texas, and Wisconsin. This run represents conditions without the proposed CAIR.
CAIR 2004_Analysis	CAIR control strategy used for much of the analytical work for the final CAIR (includes AR/DE/NJ for annual controls and no ozone season cap and is the IPM run used for air quality modeling)
CAIR 2004_Final	Final CAIR policy (includes annual and ozone season caps for the States who contribute to PM2.5 and/or ozone nonattainment)
CAIR 2004_Final_DE and NJ	CAIR Final policy with DE and NJ included in the annual program (based off CAIR 2004_Final)
BART 2004_Nationwide	Nationwide BART control strategy
CAIR + BART 2004	CAIR Analysis control strategy, with BART requirements in non-CAIR states
CAIR 2004_CSP	CAIR Final policy with annual NO _x compliance supplement pool
Base Case 2004_EIA	Base Case run with EIA assumptions for the difference between natural gas prices and coal prices, as well as EIA's projection of electricity growth
CAIR 2004_EIA	CAIR Analysis run with EIA assumptions for the difference between natural gas prices and coal prices, as well as EIA's projection of electricity growth
CAIR 2004_EIA_One Phase	CAIR Analysis run with EIA assumptions for the difference between natural gas prices and coal prices, EIA's projection of electricity growth, and Phase II caps pushed to 2010
CAIR 2004_EIA_SCR Costs	CAIR Analysis run with EIA assumptions for the difference between natural gas prices and coal prices, EIA's projection of electricity growth, and SCR capital costs and fixed O&M costs scaled up 30 percent
CAIR 2004_SCR Bypass_NOx SIP Call	CAIR Analysis run with additional SCR bypass cost for existing NO _x SIP Call units only

(Continued)

 Table D-9. Listing of Runs from the Integrated Planning Model Used in Analyses Done

 in Support of the CAIR Final Rule Analyses (continued)

Run Name	Run Description					
CAIR 2004_No NO _x	CAIR Analysis SO ₂ policy, with base case NO _x					
CAIR 2004_No SO ₂	CAIR Analysis $NO_x$ policy, with base case $SO_2$					
CAIR 2004_No SO ₂ _Summer NO _x _1	CAIR NO _x control during ozone season only in all CAIR states, with base case $SO_2$					
CAIR 2004_No SO ₂ _Summer NO _x _2	CAIR NO _x control during ozone season in 8-hour ozone states, with base case $SO_2$					
BART 2004_No NO _x	Nationwide BART SO ₂ limits, with base case $NO_x$					
BART 2004_No SO ₂	Nationwide BART $NO_x$ limits, with base case $SO_2$					
CAIR 2004_No Retirement Ratios	CAIR Analysis run but with an $SO_2$ cap rather than use of Title IV allowance retirement ratios for use in CAIR					
Parsed Files						
EPA base case parsed for year 2010						
EPA base case parsed for year 2015						
EPA base case parsed for year 2020						
EPA CAIR parsed for year 2010						
EPA CAIR parsed for year 2015						
EPA CAIR parsed for year 2020						
EPA BART parsed for year 2015						
EPA CAIR + BART parsed for year 2015						

#### **APPENDIX E**

#### CAIR INDUSTRY-SECTOR IMPACTS

EPA estimates the direct costs of implementing CAIR at \$3.6 billion in 2015 in the CAIR region. Given the impact of this rule on electricity generators, we believe it is important to gauge the extent to which the rule might affect other industry sectors. To do so, we conducted a limited analysis of the economy-wide effects of implementing CAIR.

We were particularly interested in learning how anticipated changes in electricity prices might affect industry sectors that are large electricity users. The models we employed indicated those impacts would be small, even without incorporating the beneficial economic effects of CAIR-related air quality improvements such as improved worker health and productivity. Rather, our analyses continue to show that the value of even the limited subset of CAIR benefits we were able to quantify substantially outweigh implementation costs. The degree to which projected benefits exceed projected costs would be even greater if we were able to include a number of other beneficial effects, such as a reduction in acid rain damage and lowering of nitrogen deposition.

By focusing only on cost-side spillover effects on the economy, the industry-sector impacts projected by our macroeconomic models are likely overstated, primarily because the positive market impacts of the CAIR on labor availability and productivity are excluded. In this regard, an independent panel of experts has encouraged EPA to work toward incorporating both beneficial and costly effects when modeling the economy-wide consequences of regulation. EPA is actively working to develop this capability.

Although neither model has yet been configured to include the indirect economic benefits of air quality improvements, EPA employed two distinct computable general equilibrium models to gauge the potential magnitude of the economy-wide effects of CAIR implementation costs. The first model, known as Intertemporal General Equilibrium Model (IGEM), has a long track record and was used by the Agency for the first of the two Clean Air Act Section 812 studies. The other model, called EMPAX-CGE, is currently in peer review and has the advantage of disaggregating the U.S. into multiple regions. As with all models, these tools have their respective strengths and weaknesses, and differences in data and choice of functional form imply that the models are likely to show slightly different results. Despite the differences between the models, the results of the respective analyses show similarly small impacts of CAIR on energy intensive industries. For example, production changes for the chemical manufacturing industry are estimated at -0.01 percent to -0.04 percent in 2010. Furthermore, neither model was configured to capture the beneficial economic consequences of the increased labor availability and productivity expected to result from CAIR-related air quality improvements. If these labor productivity improvements were included, the small production output decreases projected by both models might be partially or entirely offset. EPA continues to investigate the feasibility of incorporating labor productivity gains and other beneficial effects of air quality improvements in computable general equilibrium models. The individual analyses of CAIR by the two general equilibrium models follow.

### E.1 IGEM Economy-wide Analysis of CAIR: Analysis of Electric-Sector Impacts

CAIR is designed to improve air quality in nonattainment areas by achieving reductions in  $SO_2$  and/or  $NO_x$  emissions from the electric power industry in 29 eastern states and the District of Columbia.

EPA has modeled the macroeconomic impacts of the electric-sector changes expected from this rule. This analysis used the IGEM developed by Dale Jorgenson, Peter Wilcoxen, and Mun Sing Ho and maintained by Dale Jorgenson Associates. This appendix discusses the economy-wide model and the approach used to analyze the rule, along with the results of this modeling.

IGEM is a dynamic computable general equilibrium model of the U.S. economy. The model is a stylized representation of the entire economy, in which supply, demand, prices, and quantities for goods and services reach equilibria for each year of the simulation's time horizon. IGEM represents the U.S. economy as 35 distinct sectors, roughly corresponding to the two-digit levels of the North American Industry Classification System (NAICS). This level of disaggregation allows for insight into the economy-wide effects of policies that directly affect only a limited number of sectors.

The model has been used in peer-reviewed academic studies and in government, private-sector, and nonprofit policy analyses, including the first study of the Clean Air Act under Section 812. In the 812 process, the model was subjected to peer review by the EPA's SAB.

#### E.1.1 Modeling Approach

EPA believes that the best tool currently available to analyze rules and policies affecting the electric power sector is IPM developed by ICF Consulting. (For information on this model, see www.epa.gov/airmarkets/epa-ipm.) IPM is a highly disaggregated model of the electric utility industry that provides a much more detailed view of changes in the sector than would be possible in the framework of an economy-wide model such as IGEM. However, IPM currently has no representation of other sectors and cannot analyze the economy-wide impacts of the rule.

EPA has chosen to use the results of IPM regarding electric sector costs and fuel quantity changes and introduce them as inputs into IGEM. Introducing these changes as inputs causes IGEM to find new equilibria, as the relative prices of factor inputs adjust and economic agents change their demand for goods and services. A change of this nature in an economy-wide model shows that, compared to a reference case (base case), some sectors experience increased demand for their outputs, while other sectors face reduced demand. The model accounts for the overall changes in economic activity, reporting the respective impacts on the output of each of the 35 individual producing industries, as well as on consumer prices, labor supply and demand, and GDP. IGEM, therefore, complements the sector-specific IPM analysis by showing how the changes in the electric sector affect the other sectors of the economy in some detail.

Please note that this analysis only accounts for CAIR impacts to the electric power sector itself as modeled by IPM. It does not account for other economic effects, including the substantial economic benefits of reduced emissions of atmospheric pollutants. The annual benefits to which EPA can assign a dollar valuation are estimated to be \$73.3 billion in 2010 and \$60.4 billion (3 percent and 7 percent discount rates, respectively) in 2010 and \$101 billion or \$86.3 billion (3 percent and 7 percent discount rates, respectively) in 2015. There are additional benefits, including environmental benefits and some health improvements for which EPA could not estimate dollar values. See Chapter 4 for more information on benefits analyses. The dollar figures cited above and throughout this appendix are in 1999 dollars.

#### E.1.2 Modeling Methodology

Economic models have different embedded assumptions and model structures; thus, it is always challenging to link two models together. Because IGEM is an aggregate, econometrically derived model of the entire U.S. economy and IPM is a detailed, technology rich model of a single sector, these challenges are certainly present. EPA developed the methodology of linking the IPM outputs as IGEM inputs by working closely with Dale Jorgenson Associates.

The approach used in the present analysis consists of two steps. The first is to use IPM to calculate the total incremental resource costs of CAIR to the electric sector compared to a reference case. These costs include changes in capital costs, operating and maintenance (O&M) costs, and fuel costs associated with electricity generation. The incremental costs then are added to the resource cost of inputs for the IGEM electricity sector. The simulation shows that these additional costs result in changes in productivity and, hence, lower supply of and demand for electricity in the rest of the economy. For businesses, governments, and households, electricity becomes relatively more expensive, and these "consumers" adjust their purchasing behaviors, substituting away from electricity.

The second step is to account for the changes IPM projects for the coal sector. More than 90 percent of coal produced in the United States is consumed in the electric sector (DOE, 2004). EPA considers the IPM projections of coal production for the power sector to be sufficiently accurate to incorporate them into the economy-wide representation of IGEM. These impacts are introduced into IGEM by adjusting the productivity of the coal sector to account for the IPM projection of the quantity change of coal consumed. The specific percentage change to the IGEM coal-sector productivity is calculated as the percentage reduction in coal consumption projected by IPM, multiplied by the fraction of total coal output consumed by the electric sector. This adjustment changes the price of coal, and all sectors then adjust, demanding relatively less coal.

Because of the differences in underlying data, model structures, and estimated parameters between IGEM and IPM, it is not possible to match both the price and quantity changes arising in each methodology. EPA and Dale Jorgenson Associates believe that more meaningful estimates for the economy arise by explicitly matching the coal quantity impacts projected by IPM rather than IPM's projected price impacts. Furthermore, although the majority of coal produced in the United States is consumed in the electric sector, this is not the case for natural gas. Therefore, IPM projections of gas usage for the electric sector are not necessarily indicative of the economy-wide demand for this fuel, and EPA has chosen not to model the impacts of CAIR on natural gas using the same approach as that used for coal. Rather, IGEM is allowed to solve for all changes in natural gas markets as the economy responds to the rule's impacts on electricity and coal markets.

#### E.1.3 Projected Impacts on Specific Industries

The productivity changes incurred by the electric sector and coal sector contribute to changes in output for virtually all sectors. This happens because, as noted above, relative price levels for electricity and for energy sources which may substitute for electricity change under CAIR. In this simulation, consumers demand for energy intensive goods tends, in general, to fall slightly, while demand for other goods tends to rise.

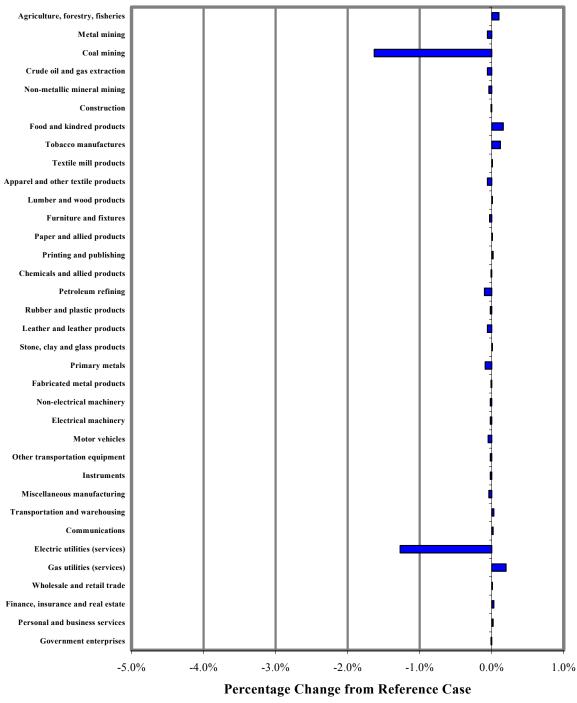
For the vast majority of the 35 IGEM sectors, the impact is less than 0.05 percent (five one hundredths of 1 percent) of forecasted domestic output measured in sales and may be positive, representing increased production, or negative, representing loss of output.

For some sectors, however, the results are more pronounced. IGEM shows increases in natural gas output, because natural gas is substituted for coal and electricity; this increase is 0.16 percent in 2010 and 0.20 percent in 2015. Other sectors benefit as well, as consumers substitute away from energy consumption to other products, particularly agriculture and food.

Sectors that face relatively larger output changes also include the electric and coal sectors. The loss in the coal sector is very similar to that predicted by IPM, at 1.27 percent in 2010 and 1.63 percent in 2015. The electric sector itself faces a loss of output of 1.01 percent in 2010 and 1.27 percent in 2015. Other energy-intensive industries also lose output, ranging from 0.01 percent for the chemical industry to 0.10 percent for petroleum refining. Figure E-1 details the 2015 results. (The complete results for 2010 and 2015 for each of the 35 sectors represented in IGEM are included later in this appendix in Section E.1.9.)

#### E.1.4 Projected Impacts on Consumer Prices

Indices of consumer prices are well-accepted measures of price levels within an economy. The indices increase when consumers face higher prices for the goods they purchase. These indices are calculated as weighted-average prices for particular "baskets" of goods commonly purchased by households. Within IGEM, "the basket" is based on household purchases of the 35 commodities available in its various markets for goods and services. The electric sector impacts of the CAIR have effects on IGEM's aggregate consumer price level. There is a direct effect as electricity becomes relatively more expensive and an indirect effect as industries that consume electricity face a higher price for



Source: Intertemporal General Equilibrium Model



a factor input, and then pass on that price increase by charging higher prices for goods and services. Figure E-2 shows the percentage change in consumer prices (approximately 0.032 percent in 2010 and 0.036 percent 2015) over the reference case.

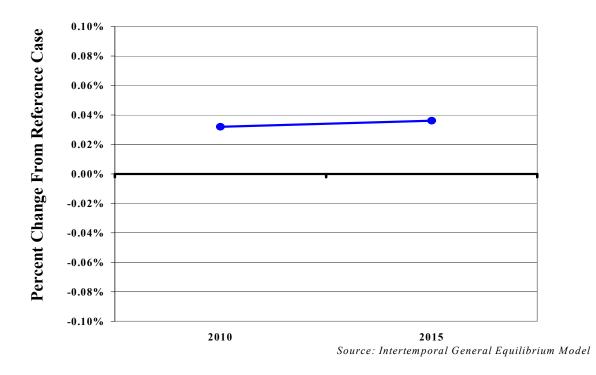


Figure E-2. Change in Consumer Prices Compared to Reference Case

### E.1.5 Projected Impacts on Labor Markets

IGEM projects changes in labor utilization under the CAIR scenario (see Figure E-3). Note that this is not an explicit statement about employment or jobs, as the IGEM model does not calculate estimates of employment or job creation. Rather, the model accounts for labor supply and demand in terms of "hours." An increase in "labor input" associated with the CAIR implies an increase in the number of quality-adjusted hours supplied by households and demanded by employers. In the IGEM framework, as energy prices rise, firms substitute other inputs, which include capital, materials, and labor. For details on the representation of labor within IGEM, and information on interpreting the results of the model, please see Section E.1.8. The change in labor input is less than 0.01 percent in 2010 and 2015.

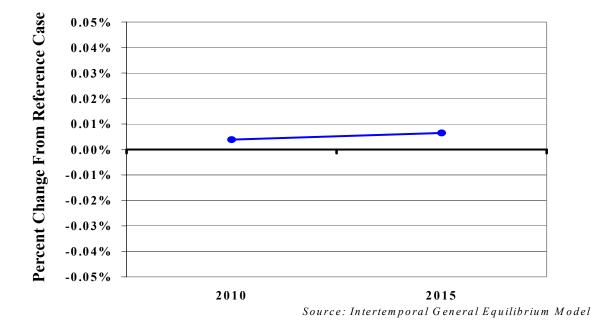


Figure E-3. Change in Labor Input Compared to Reference Case

#### E.1.6 Projected Impacts on GDP

In this analysis, the costs incurred by the electric sector contribute to a change in GDP. EPA believes that these GDP results are incomplete, as they do not represent an economy-wide modeling of the substantial economic benefits expected to accrue due to decreased health impacts and other effects of reduced emissions. Because these results are incomplete, the impacts on GDP should not be construed as the costs of the rule. The results here show only the impact of resource cost changes to the electricity and coal sectors. These changes cause the other IGEM sectors and households to adjust to accommodate higher energy prices, and the economy incurs net losses. Almost all general equilibrium models can be expected to show that a change in resource costs to one sector tends to be transmitted to other sectors. IGEM is no exception. As shown in Figure E-4, IGEM projects the overall cost as a result of the direct effects on coal and electricity to be approximately 0.03 percent of GDP (three one hundredths of one percent) in 2010 and 2015.

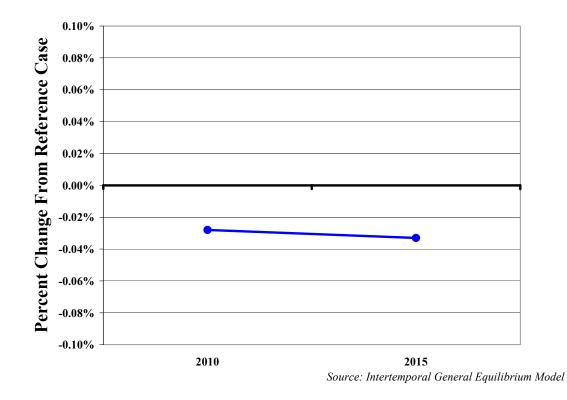


Figure E-4. Change in Gross Domestic Product (GDP) Compared to Reference

However, these effects must be viewed in the context of a growing economy; IGEM projects overall reference case GDP growth to be similar to that projected by the Energy Information Administration's 2004 Annual Energy Outlook (AEO). According to IGEM, U.S. GDP is projected to grow to \$13.9 trillion in 2010 and to \$16.2 trillion in 2015. Figure E-5 shows the projected growth of GDP in the base case compared to the growth of GDP including the costs (but not the benefits) of CAIR. The dotted line representing growth in the economy in the scenario which includes CAIR impacts is slightly lower than the solid line representing the reference case. (Note that in Figure E-5, the apparent difference is smaller than thickness of the lines. The policy line appears to lie nearly on top of the reference line).

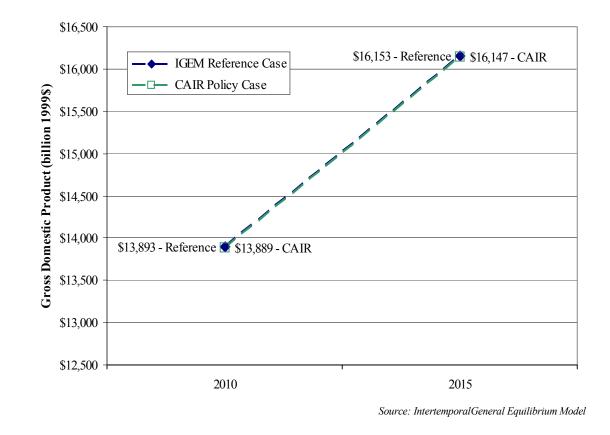


Figure E-5. U.S. Gross Domestic Product (GDP): Reference Case vs. CAIR

# E.1.7 IGEM Model Description

As noted above, IGEM is a dynamic, multiperiod, multisector computable general equilibrium model. IGEM solves for supply and demand equilibria in the markets for 35 commodities produced by 35 domestic industries as well as imported from overseas. These commodities are demanded by producers as intermediate inputs to production or by households, businesses, government, or exporters as finished goods. Each producing sector is represented by equations that specify the sector's production (outputs) and factor demands (inputs) and the degree to which these factor inputs might be substituted for one another. IGEM is unique among models of this type in that the important economic parameters used in the model were derived entirely from econometric analyses of historical U.S. economic data. These parameters include measures of the substitutability of inputs to production, consumers' relative demands for goods and services, and trends in productivity improvement.

IGEM is characterized by "perfect foresight," in which economic agents act rationally with complete knowledge of the future to maximize the long-term net benefits arising from their decisions. It is also important to note that IGEM represents capital as "perfectly mobile," so that infrastructure is easily converted to other uses as dictated by demand considerations within the context of overall availability (supply).

# E.1.8 Labor Representation in IGEM

IGEM is a "full employment" model, in which all inputs are fully used, including the labor market. This assumption follows from IGEM's focus on market equilibria (supply and demand balances) in all markets, including labor, and from the fact that labor is represented by quality-adjusted hours and not by persons. Labor supply is endogenously determined in the model, so that the amount of labor supply and demand and the real (i.e., inflation-adjusted) wage rate adjust in response to policy changes. Labor use, the labor variable reported by IGEM, is a proxy measurement for "employment," but there are distinct differences. In the full employment framework, households decide the quantity of labor services to provide. In IGEM, workers have the option to enjoy leisure or provide labor and do so based on the prevailing real wage rate. An increase in the labor utilization rate implies that individuals are choosing to provide more labor to the market in response to firms' demand for labor as a substitute for electricity and coal.

# E.1.9 Sector Detail

Thirty-five sectors comprise the U.S. economy and are represented in IGEM. The list of these sectors, along with the response of the sectors to the costs of CAIR, are presented in Table E-1. The table shows the percentage change in output from each sector (the change is in "constant – dollar sales" as a measure of output).

# E.2 EMPAX-CGE Regional Macroeconomic Analysis of CAIR

CAIR reduces emissions of  $SO_2$  and  $NO_x$  from electricity generation to improve air quality.¹ To complement the analysis of CAIR effects on electricity generation conducted by the EPA using the IPM,² the macroeconomic implications of this rule have been estimated

¹See <http://www.epa.gov/cleanair2004/> for details.

²See <http://www.epa.gov/airmarkt/epa-ipm/> for complete IPM documentation.

# Table E-1. Change in Sector Output in Response to CAIR

Sector	Percent Change from Reference		
	2010	2015	
Agriculture, forestry, fisheries	0.07%	0.10%	
Metal mining	-0.04%	-0.06%	
Coal mining	-1.27%	-1.63%	
Crude oil and gas extraction	-0.04%	-0.06%	
Non-metallic mineral mining	-0.03%	-0.04%	
Construction	-0.01%	-0.01%	
Food and kindred products	0.11%	0.16%	
Tobacco manufactures	0.08%	0.12%	
Textile mill products	-0.01%	0.01%	
Apparel and other textile products	-0.05%	-0.06%	
Lumber and wood products	0.00%	0.01%	
Furniture and fixtures	-0.02%	-0.03%	
Paper and allied products	0.01%	0.01%	
Printing and publishing	0.01%	0.02%	
Chemicals and allied products	-0.01%	-0.01%	
Petroleum refining	-0.07%	-0.10%	
Rubber and plastic products	-0.02%	-0.02%	
Leather and leather products	-0.05%	-0.06%	
Stone, clay and glass products	0.00%	0.01%	
Primary metals	-0.07%	-0.09%	
Fabricated metal products	-0.01%	-0.01%	
Non-electrical machinery	-0.02%	-0.02%	
Electrical machinery	-0.02%	-0.02%	
Motor vehicles	-0.03%	-0.05%	
Other transportation equipment	-0.01%	-0.02%	
Instruments	-0.03%	-0.02%	
Miscellaneous manufacturing	-0.03%	-0.04%	
Transportation and warehousing	0.02%	0.03%	
Communications	0.02%	0.02%	
Electric utilities (services)	-1.01%	-1.27%	
Gas utilities (services)	0.16%	0.20%	
Wholesale and retail trade	0.01%	0.01%	
Finance, insurance and real estate	0.03%	0.03%	
Personal and business services	0.02%	0.02%	
Government enterprises	0.00%	-0.01%	

Source: Intertemporal General Equilbrium Model

using EPA's EMPAX-CGE model. EMPAX-CGE is a macroeconomic simulation model developed by RTI International (RTI) for EPA's Office of Air Quality Planning and Standards (OAQPS). The focus of this analysis of CAIR is examining the sectoral and regional distribution of economic effects across the U.S. economy. This appendix section discusses the EMPAX model, the approach used to incorporate electricity-sector results from IPM, and the results of the macroeconomic analysis. Please note that this analysis focuses on electricity-sector impacts of CAIR as estimated by IPM. It does not account for other economic and noneconomic effects, especially the substantial economic and health benefits associated with reduced emissions.

# E.2.1 Background and Summary of EMPAX-CGE Model³

EMPAX was first developed in 2000 to support the economic analysis of EPA's maximum achievable control technology (MACT) rules controlling emissions from three categories of combustion sources (reciprocating internal combustion engines, boilers, and turbines). The initial framework consisted of a national multimarket partial-equilibrium model with linkages between manufacturing industries and the energy sector. Effects of combustion rules on these industries were estimated through their influence on energy prices and output. Modified versions of EMPAX were subsequently used to analyze economic impacts of strategies for improving air quality in the Southern Appalachian mountain region.

Recent work on EMPAX has extended its scope to cover all aspects of the U.S. economy at a regional level in either static or dynamic modes (the dynamic version of EMPAX is used in this analysis). Although major regulations directly affect a large number of industries, substantial indirect impacts can also result from changes in production, input use, income, and household consumption patterns. Consequently, EMPAX now includes economic linkages among all industrial and energy sectors as well as households that supply factors of production and purchase goods (i.e., a computable general equilibrium, or CGE, framework). This gives the version of EMPAX called EMPAX-CGE the ability to trace economic impacts as they are transmitted throughout the economy and allows it to provide critical insights to policy makers evaluating the magnitude and distribution of costs associated with environmental policies. The EMPAX-CGE model was used to investigate macroeconomic impacts of the Interstate Air Quality Rule, predecessor to CAIR.⁴

³See Section E.2.8 for additional details on the EMPAX-CGE model.

⁴See <http://www.epa.gov/interstateairquality/technical.html>.

The dynamic version of EMPAX-CGE employed in this analysis of CAIR is an intertemporally optimizing model. Agents have perfect foresight and maximize utility across all time periods subject to budget constraints, while firms maximize profits subject to technology constraints. Nested constant elasticity of substitution (CES) functions are used to portray substitution possibilities available to producers and consumers. Along with the underlying data, the nesting structures and associated substitution elasticities define current production technologies and possible alternatives. Most industries have constant returns to scale with the exception of fossil-fuel and agriculture industries that have decreasing returns to scale due to use of factors in fixed supply (land and inputs of primary fuels, respectively). The current tax structure of the United States is included in EMPAX-CGE to account for "tax interaction" effects, where interactions between tax distortions and environmental policies can affect macroeconomic costs of policies.

The economic data in this CGE model come from state-level information provided by the Minnesota IMPLAN Group, and the energy data come from the DOE's Energy Information Agency (EIA). In the dynamic version of EMPAX-CGE, these data are used to define five regions within the United States, each containing 17 industries and four types of households classified by income.⁵ The five regions have been selected to preserve important regional differences in electricity generation technologies, and 17 industries are included that cover five important types of energy (coal, crude oil, electricity from fossil and nonfossil generation, natural gas, and refined petroleum), the energy-intensive industries most likely to be affected by environmental policies, and the remaining sectors of the economy.

Four sources of economic growth are included: technological change from improvements in energy efficiency, growth in the available labor supply from population growth and changes in labor productivity, increases in stocks of natural resources, and capital accumulation. Changes in energy use per unit of output are modeled through exogenous autonomous energy efficiency improvements (AEEI). The baseline solution in EMPAX-CGE matches, as closely as possible, EIA forecasts for energy production by fuel type, energy prices, fuel consumption by industry, industrial output, and regional economic growth through 2025.⁶

⁵Static versions of EMPAX-CGE have more industries and households because they do not have to solve for multiple time periods simultaneously and, consequently, have few computational constraints on the number of industries and households.

⁶EIA forecasts from the Annual Energy Outlook 2003 (AEO) are used in this analysis.

Distortions associated with the existing tax structure in the United States have been included in EMPAX-CGE. A wide range of theoretical and empirical literature has examined "tax interactions" and found that they can substantially alter costs of environmental (and other) policies. The IMPLAN economic database used by EMPAX-CGE includes information on some types of taxes, which have been combined with other sources to cover important distortions from capital and income taxes.

# E.2.2 Modeling Approach for Electricity Policies

EMPAX-CGE can be used to analyze a wide array of policy issues and is capable of estimating how a change in a single part (or multiple parts) of the economy will influence producers and consumers across the United States. However, although CGE models have been used extensively to analyze climate policies that limit carbon emissions from electricity production,⁷ some other types of utility emissions policies are more difficult to consider. Unlike carbon dioxide, emissions of pollutants such as SO₂, NO_x, and mercury are not necessarily proportional to fuel use.

These types of emissions can be lowered by a variety of methods: fuel switching from high- to low-sulfur coal, moving from coal- to gas-fired generation, and/or installing retrofit equipment designed to reduce emissions. However, the boiler-specific nature of these decisions, and their costs and effects, cannot be adequately captured by the more general structure of a CGE model. In addition, because of the ways that retrofits and construction of new generating units affect electricity prices and fuel use, a detailed characterization of electricity markets is preferable when estimating implications of policies like CAIR. For these reasons, we developed an interface that allows a linkage between EMPAX-CGE and the IPM model.

IPM is a comprehensive model of electricity generation and transmission in the United States. The model contains data on all generating units available to dispatch electricity to the national grid, their existing equipment configurations and fuel consumption, transmission constraints, and generating costs. It includes characteristics of new units and retrofits that can be built and/or installed. IPM is capable of estimating how electric utilities will respond to policies by determining the least-cost methods of generating sufficient electricity to meet demands, while meeting emissions reduction (and other) objectives.

⁷See, for example, the analyses of energy/climate using CGE models organized by the Stanford University Energy Modeling Forum (http://www.stanford.edu/group/EMF/home/index.htm).

(disaggregated unit-level analyses of electricity policies) with the strengths of CGE models (macroeconomic effects of environmental policies) allows investigation of economy-wide implications of policies that would normally be hard to estimate consistently and effectively. For electricity-generation regulations like CAIR that require a very disaggregated level of analysis, IPM can determine for EMPAX-CGE a number of electricity market outcomes needed to evaluate macroeconomic implications of policies. The linkage with IPM then allows EMPAX-CGE to take these findings and use them in "counterfactual" policy evaluations. Among the many results provided by IPM, several can potentially have significant implications for the rest of the economy including changes in electricity prices, fuel consumption by utilities, fuel prices, and changes in electricity production expenditures. EMPAX-CGE is capable of simultaneously incorporating some or all of these IPM findings. depending on the desired type and degree of linkage between the two models. At the regional level, EMPAX-CGE can match changes estimated by IPM for the following variables: electricity prices (percentage change in retail prices) coal and gas consumption for electricity (percentage changes in Btus)

- coal and gas prices (percentage changes in prices)
- coal and gas expenditures (\$ changes—Btus of energy input times \$/MMBtu)

However, IPM does not fully consider how changes in the electricity sector, or electricity prices, will affect the rest of the U.S. economy. Combining the strengths of IPM

- capital costs (\$ changes)
- fixed operating costs (\$ changes)
- variable operating costs (\$ changes)

For EMPAX-CGE to effectively incorporate IPM data on changes in costs, they have to be expressed in terms of the productive inputs used in CGE models (i.e., capital, labor, and material inputs produced by other industries). Rather than assume these costs represent a proportional scaling up of all inputs to the electricity industry in EMPAX-CGE, we use Nestor and Pasurka (1995) data on purchases made by industries for environmental protection reasons to allocate these additional expenditures across inputs within EMPAX-CGE. Once these expenditures are specified, the incremental costs from IPM can be used to adjust the production technologies and input purchases by electricity generation in the CGE model.

# E.2.3 Modeling Methodology for CAIR

The macroeconomic impacts of CAIR, as simulated by CGE models, will be a function of the methodology used to link IPM to EMPAX-CGE and the economic interactions accounted for by the CGE model. Initial effects will revolve around how using additional resources in electricity generation draws some capital, labor, and materials from other sectors of the economy. This, in turn, may affect prices in markets supplying these inputs to utilities. Similarly, changes in coal and gas use in electricity and associated impacts on their prices will have implications for the rest of the economy (although any spillovers in coal markets will have limited effects outside of electricity because most is used for generation). In addition, any electricity price increases associated with these initial effects will encourage improvements in energy efficiency, switching to alternate forms of energy (increases in natural gas prices may mitigate this effect), and lower consumption of electricity in general (these demand decreases would lead to lower production levels with associated benefits for the environment). The magnitude of these adjustments will be a function of the structure of the CGE model and the elasticities in it that control the ease of these substitutions.

The macroeconomic effects beyond energy production and consumption decisions also depend on the theoretical structure of the CGE model used in the analysis. Similar to the perfect-foresight nature of IPM, CGE models like EMPAX-CGE assume that firms and consumers will observe and anticipate policies to be enacted in the future. This causes them to adjust their behavior and investment decisions in all time periods in the model (including the starting year of the model). As a result, anticipation of changes in production and consumption costs in the future will cause shifts in behavior in all model years as people prepare ahead of policy enactment. The aggregate implications of these changes will also be influenced by income effects (how people will alter consumption levels in response to having more or less money) and substitution effects (how people will alter their patterns of consumption purchases in response to changes in relative prices of goods). For example, an anticipated decrease in labor productivity in the future (leading to lower wages) may cause an increase in work effort today, while labor is more productive. Alternatively, it may lead

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to a decrease in work effort today because, in part, labor today is used to generate capital goods for tomorrow, which will be used to augment less-productive labor in the future.

The methodology used to link IPM and EMPAX-CGE for this CAIR analysis focuses on CAIR resource costs and implications for coal use by utilities.⁸ IPM estimates of additional resources used by electric utilities (the capital, fixed, and variable costs) are used to adjust generation technologies in EMPAX-CGE. The same procedure is used to account for incremental increases in natural gas purchases by utilities (although all these costs are applied exclusively to natural gas inputs to electricity generation in EMPAX-CGE, rather than being apportioned using the Nestor and Pasurka data). Given that approximately 90 percent of all coal is consumed in generation, we use IPM estimates of changes in coal use directly within EMPAX-CGE, expressed as percentage changes in Btus, rather than adopting a less direct linkage. However, for both coal and natural gas, EMPAX-CGE is allowed to estimate the impacts on commodity prices faced by the rest of the economy.

### E.2.4 Projected Impacts on Specific Industries

Impacts of CAIR on electricity-generation costs and their subsequent effects on electricity prices, along with coal market changes, will affect output and prices of all industries in EMPAX-CGE. These effects may increase or decrease output and/or revenue, depending on their implications for production costs and technologies, and shifts in household demands. However, as shown in Figure E-6, estimates for output changes from CAIR (outside of electricity and coal) are generally around 0.05 percent and may be positive or negative.

Some industries are affected more than others and others may increase production. Natural gas output increases as electric utilities switch out of coal generation and as higher electricity prices cause other businesses and consumers to move to alternate energy sources. Energy-intensive sectors of the economy are relatively more affected than other firms because they rely more heavily on electricity and other fuels. However, the largest of these declines in output (aluminum) is approximately two-tenths of 1 percent.

⁸See Section E.2.15 for EMPAX-CGE results using other linkages to IPM.

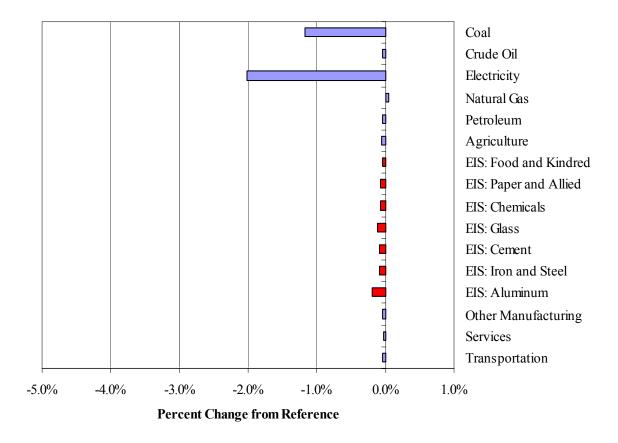
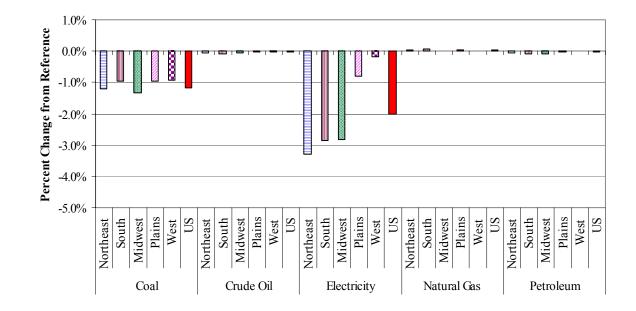


Figure E-6. CAIR Impacts on U.S. Domestic Output, 2015

Source: EMPAX-CGE.

Regional effects tend to show variation that does not appear at the national level. Figure E-7 shows these regional results for energy markets and highlights the aggregate U.S. results with a solid bar. The largest differences are in electricity generation because the western part of the United States is relatively unaffected by the policy, other than through spillover effects reflected in both IPM and EMPAX-CGE. Increases in natural gas output and declines in coal production are also distributed unevenly across the country.



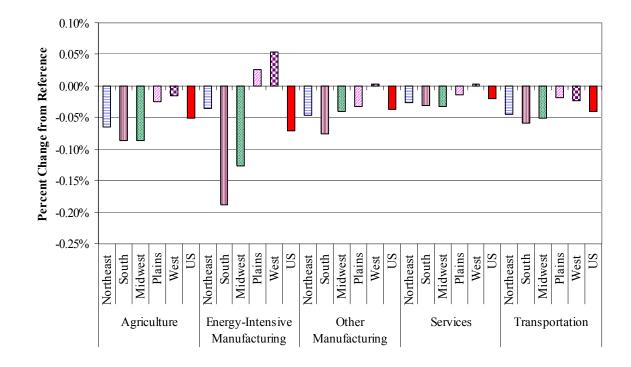
# Figure E-7. CAIR Impacts on Regional Energy Output, 2015

Source: EMPAX-CGE.

Figure E-8 shows regional results for nonenergy industries in EMPAX-CGE.⁹ Although these sectors show considerable regional variation based on differences in production methods and energy price effects, the majority of the impacts are less than five one-hundredths of 1 percent. Energy-intensive firms in the West experience a comparative advantage relative to other parts of the country. Impacts on the services industry are uniformly small, however; given the size of this sector, the effect on total revenue is larger than for all other industries combined.

Although the average effect on energy-intensive sectors industries is negative because of increased energy input costs, industries in some parts of the United States are estimated to be raising their output. Even though energy prices have risen slightly, they

⁹Results for the seven components of energy-intensive sectors are aggregated in Figure E-8 for presentation purposes, although they are all included simultaneously in the EMPAX-CGE model run.



# Figure E-8. CAIR Impacts on Regional Industrial Output, 2015

Source: EMPAX-CGE.

experience an advantage over similar firms in other regions that face proportionately larger price increases. Although the West sees the greatest improvement in comparative advantage, output also rises in other regions (see Figure E-9).

# E.2.5 Projected Impacts on Consumer Prices

Changes in consumer price levels are used to measure price effects of policies and any resulting implications for average purchase prices paid by households. EMPAX-CGE calculates an overall price level across the "basket" of goods and services bought by consumers. For a policy like CAIR, consumer price levels will be affected directly by changes in electricity prices faced by households and indirectly by changes in goods prices that have been produced using electricity. Figure E-10 shows percentage changes in average consumer prices on average ranging from 0.02 to 0.04 percent.

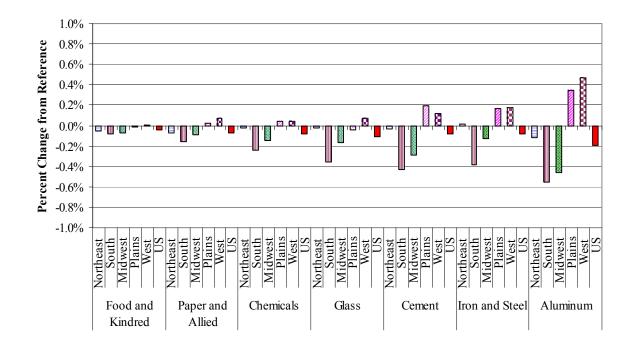


Figure E-9. CAIR Impacts on Regional Energy-Intensive Output, 2015

### E.2.6 Projected Impacts on Labor Markets

CGE models like EMPAX-CGE typically consider how policies may influence labor markets through how they alter the number of productivity-adjusted hours of labor supplied by households (this is not the same as estimating jobs or employment). Empirical estimates of labor-supply elasticities are used by EMPAX-CGE to simulate how demands by firms and supply decisions by households are made, along with resulting implications for real wages. EMPAX-CGE, similar to IGEM, is a full-employment model in which households choose between labor and leisure time, based on both income and substitution effects.

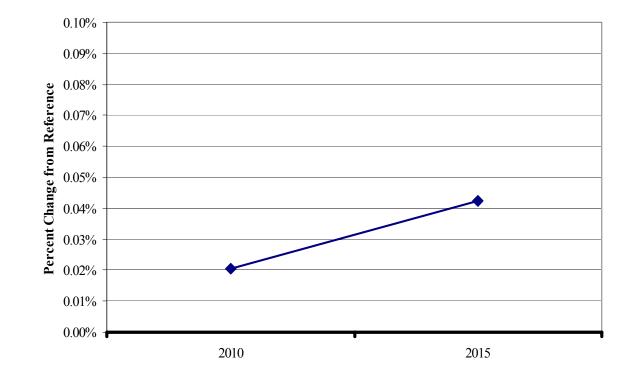


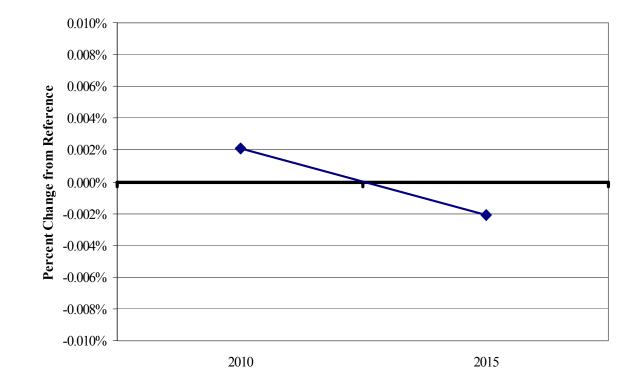
Figure E-10. Change in Consumer Prices Compared to Reference Case

Source: EMPAX-CGE.

Figure E-11 gives EMPAX-CGE's projected impacts of CAIR on labor markets. The results indicate that people are choosing to work slightly fewer hours in response to small declines in real wage rates, rather than work more hours to offset additional costs of purchasing goods. All of these effects are extremely small, however, on the order of two one-thousandths of 1 percent.

# E.2.7 Projected Impacts on GDP

The combination of all economic interactions as described earlier will be reflected in the changes in GDP estimated by a CGE model. Given that this cost-based approach to analyzing CAIR does not reflect its benefits to the environment, public health, and labor productivity, CGE models (including EMPAX-CGE) will tend to estimate declines in total

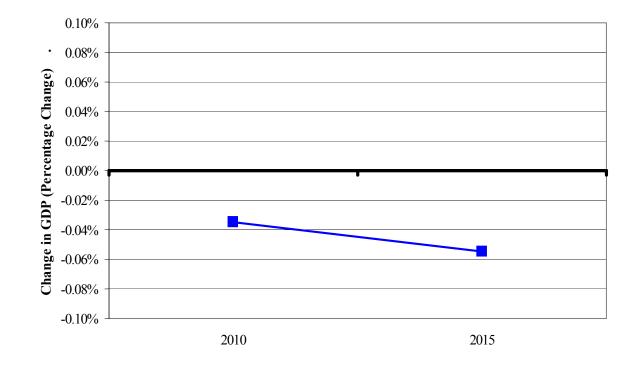


# Figure E-11. Change in Labor Inputs Compared to Reference Case

Source: EMPAX-CGE

production in the United States, as shown in Figure E-12. Because these results are incomplete and do not reflect potential benefits of CAIR, the impacts on GDP should not be construed as the costs of the rule. EMPAX-CGE projects decreases in GDP of between 0.03 percent and 0.05 percent (five one-hundredths of 1 percent).

Overall, it should be noted that the estimated implications of CAIR for U.S. GDP are extremely small relative to the total size of the economy. Figure E-13 illustrates GDP in the model baseline and CAIR policy cases. As shown, the GDP impact is negligible and, in fact, it is not possible to adjust the scale of the graph to the point where the two lines do not overlap. Even these small costs could be reversed if the CGE analyses were extended to include benefits associated with CAIR such as improvements in labor productivity from environmental improvements.

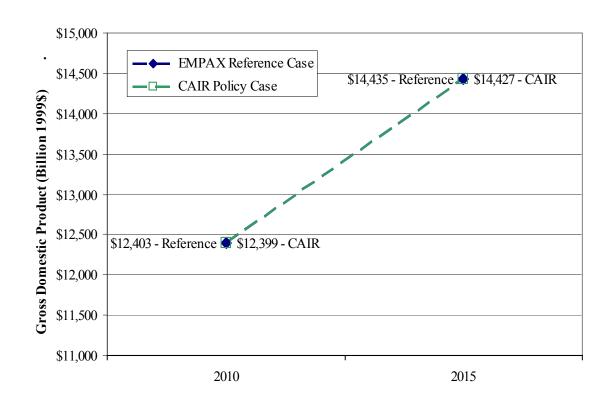


# Figure E-12. Change in GDP Compared to Reference Case

Source: EMPAX-CGE

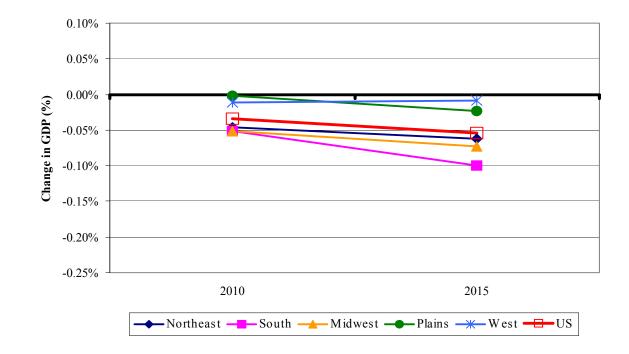
National GDP effects like those in Figure E-13 may tend to obscure variation at a regional or local level. Several potential sources of divergences in regional impacts exist:

- differences in IPM regional results based on regional mixes of generation technologies (coal, gas, oil, and nonfossil use), which may be averaged out at a national level;
- differences in regional production and consumption patterns for electricity and nonelectricity energy goods;
- differences in industrial composition of regional economies;
- differences in household consumption patterns; and
- differences in regional growth forecasts.



**Figure E-13. U.S. Gross Domestic Product (GDP): Reference Case vs. CAIR** Source: EMPAX-CGE.

Figure E-14 presents the regional GDP changes estimated by EMPAX-CGE that underlie the national U.S. results above. As with other types of results, western states are relatively unaffected by CAIR for several reasons: most of the resource costs are experienced by generators on the East Coast and in the Midwest, the industrial composition of the West (especially California) tends to lean towards less energy-intensive industries like services, and production patterns for energy-intensive sectors shift towards the West as it experiences an improvement in its comparative advantage in their production. Other parts of the United States, like the South and Midwest, depend more on coal-fired generation than the West and, as a result, have slightly larger relative GDP declines than the U.S. average. In the Northeast, there is a relatively equal reliance on coal versus natural gas in electricity production, so its estimated impacts are in line with national averages.



# Figure E-14. Change in Regional GDP Compared to Reference Case

Source: EMPAX-CGE.

Tables E-2 and E-3 show EMPAX-CGE estimates of changes in revenue and output quantities for 2010 and 2015.

# E.2.8 EMPAX-CGE Model Description: General Model Structure

This section provides additional details on the EMPAX-CGE model structure, data sources, and assumptions. The version of EMPAX-CGE used in this CAIR analysis is a dynamic, intertemporally optimizing model that solves in 5-year intervals from 2005 to 2050. It uses the classical Arrow-Debreu general equilibrium framework wherein households maximize utility subject to budget constraints, and firms maximize profits subject to technology constraints. The model structure, in which agents are assumed to have perfect foresight and maximize utility across all time periods, allows agents to modify behavior in anticipation of future policy changes, unlike dynamic recursive models that assume agents do not react until a policy has been implemented.

Model Run	Industry	2010	2015
Percentage Change in Revenue (%)	Coal	-2.73%	-3.13%
	Crude oil	-0.04%	-0.08%
	Electricity	0.60%	0.93%
	Natural gas	0.38%	0.16%
	Petroleum	-0.03%	-0.06%
	Agriculture	-0.04%	-0.07%
	Energy-intensive manufacturing	-0.04%	-0.07%
	Other manufacturing	-0.03%	-0.07%
	Services	-0.04%	-0.06%
	Transportation	-0.04%	-0.09%
Percentage Change in Quantity (%)	Coal	-0.81%	-1.17%
	Crude oil	-0.02%	-0.04%
	Electricity	-1.33%	-2.01%
	Natural gas	0.08%	0.05%
	Petroleum	-0.02%	-0.04%
	Agriculture	-0.03%	-0.05%
	Energy-intensive manufacturing	-0.04%	-0.07%
	Other manufacturing	0.00%	-0.04%
	Services	-0.01%	-0.02%
	Transportation	-0.01%	-0.04%

# Table E-2. U.S. Domestic Output Changes

Source: EMPAX-CGE.

Model Run	Industry	2010	2015
Percentage Change in Revenue (%)	Food and Kindred	-0.04%	-0.06%
	Paper and Allied	-0.03%	-0.07%
	Chemicals	-0.04%	-0.08%
	Glass	-0.04%	-0.09%
	Cement	-0.01%	-0.02%
	Iron and Steel	-0.02%	-0.06%
	Aluminum	0.02%	-0.02%
Percentage Change in Quantity (%)	Food and Kindred	-0.03%	-0.04%
	Paper and Allied	-0.03%	-0.07%
	Chemicals	-0.04%	-0.08%
	Glass	-0.05%	-0.11%
	Cement	-0.02%	-0.09%
	Iron and Steel	-0.02%	-0.08%
	Aluminum	-0.12%	-0.20%

Table E-3. U.S. Domestic Energy-Intensive Sector Output Changes

Source: EMPAX-CGE.

Nested CES functions are used to portray substitution possibilities available to producers and consumers. Figure E-15 illustrates this general framework and gives a broad characterization of the model.¹⁰ Along with the underlying data, these nesting structures and associated substitution elasticities determine the effects that will be estimated for policies. These nesting structures and elasticities used in EMPAX-CGE are generally based on the Emissions Prediction and Policy Analysis (EPPA) Model developed at the Massachusetts Institute of Technology (Babiker et al., 2001). Although the two models are quite different (EPPA is a recursive-dynamic, international model focused on national-level climate-change policies), both are intended to simulate how agents will respond to environmental policies.

¹⁰Although it is not illustrated in Figure E-15, some differences across industries exist in their handling of energy inputs. In addition, the agriculture and fossil-fuel sectors in EMPAX-CGE contain equations that account for the presence of fixed inputs to production (land and fossil-fuel resources, respectively).

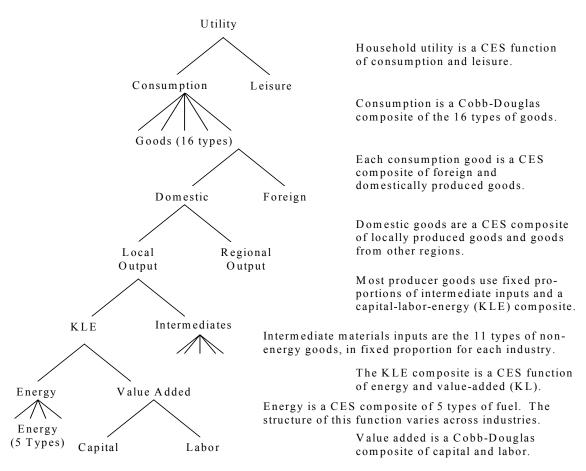


Figure E-15. General Production and Consumption Nesting Structure in EMPAX-CGE

Given this basic similarity, EMPAX-CGE has adopted a comparable structure. EMPAX-CGE is programmed in the GAMS¹¹ language (Generalized Algebraic Modeling System) and solved as a mixed complementarity problem (MCP)¹² using MPSGE software (Mathematical Programming Subsystem for General Equilibrium).¹³ The PATH solver from GAMS is used to solve the MCP equations generated by MPSGE.

¹¹See Brooke et al. (1998) for a description of GAMS (http://www.gams.com/).

¹²Solving EMPAX-CGE as a MCP problem implies that complementary slackness is a feature of the equilibrium solution. In other words, any firm in operation will earn zero economic profits and any unprofitable firms will cease operations. Similarly, for any commodity with a positive price, supply will equal demand, or conversely any good in excess supply will have a zero price.

¹³See Rutherford (1999) for MPSGE documentation (http://debreu.colorado.edu).

#### E.2.9 Data Sources

The economic data come from state-level information provided by the Minnesota IMPLAN Group¹⁴ and energy data come from EIA.¹⁵ Although IMPLAN data contain information on the value of energy production and consumption in dollars, these data are replaced with EIA data for several reasons. First, the policies being investigated typically focus on energy markets, making it essential to include the best possible characterization of these markets in the model. Although the IMPLAN data are developed from a variety of government data sources at the U.S. Bureau of Economic Analysis and U.S. Bureau of Labor Statistics, these data do not always agree with energy information collected by EIA directly from manufacturers and electric utilities. Second, it is necessary to have physical quantities for energy consumption in the model to portray effects of environmental policies. EIA reports physical quantities, while IMPLAN does not. Finally, although the IMPLAN data reflect the year 2000, the initial baseline year for the model is 2005. Thus, AEO energy production and consumption, output, and economic growth forecasts for 2005 are used to adjust the year 2000 IMPLAN data.

EMPAX-CGE combines these economic and energy data to create a balanced social accounting matrix (SAM) that provides a baseline characterization of the economy. The SAM contains data on the value of output in each sector, payments for factors of production and intermediate inputs by each sector, household income and consumption, government purchases, investment, and trade flows. A balanced SAM for the year 2005 consistent with the desired sectoral and regional aggregation is produced using procedures developed by Babiker and Rutherford (1997) and described in Rutherford and Paltsev (2000). The methodology relies on standard optimization techniques to maintain the calculated energy statistics while minimizing the changes needed in the economic data to create a new balanced SAM that matches AEO forecasts for the baseline model year of 2005.

These data are used to define 10 regions within the United States, each containing 40 industries. Regions have been selected to capture important differences across the country in electricity-generation technologies, while industry aggregations are controlled by available energy consumption data. Prior to solving EMPAX-CGE, these regions and industries are aggregated up to the categories to be included in the analysis.

¹⁴See <http://www.implan.com/index.html> for a description of the Minnesota IMPLAN Group and its data.

¹⁵These EIA sources include AEO 2003, the Manufacturing Energy Consumption Survey, State Energy Data Report, State Energy Price and Expenditure Report, and various annual industry profiles.

Table E-4 presents the industry categories included in EMPAX-CGE for the CAIR analysis. Their focus is on maintaining as much detail in the energy-intensive sectors¹⁶ as is allowed by available energy consumption data and computational limits of dynamic CGE models. In addition, the electricity industry is separated into fossil-fuel generation and nonfossil generation, which is necessary because many electricity policies like CAIR affect only fossil-fired electricity.

EMPAX Industry	NAICS Classifications	
Coal	2121	
Crude Oil ^a	211111	
Electricity (fossil and nonfossil)	2211	
Natural Gas	211112, 2212, 4862	
Petroleum Refining	324	
Agriculture	11	
Energy-Intensive Sector: Food	311	
Energy-Intensive Sector: Paper and Allied	322	
Energy-Intensive Sector: Chemicals	325	
Energy-Intensive Sector: Glass	3272	
Energy-Intensive Sector: Cement	3273	
Energy-Intensive Sector: Iron and Steel	3311	
Energy-Intensive Sector: Aluminum	3313	
Other Manufacturing	312-316, 321, 323, 326-327, 331-339	
Services	All Others	
Transportation ^b	481-488	

# Table E-4. EMPAX-CGE Industries

^a Although NAICS 211111 covers crude oil and gas extraction, the gas component of this sector is moved to the Natural Gas industry.

^b Transportation does not include NAICS 4862 (natural gas distribution), which is part of the natural gas industry.

¹⁶Energy-intensive sectors industry categories are based on EIA definitions of energy-intensive manufacturers in the *Assumptions for the Annual Energy Outlook 2003*.

Figure E-16 shows the five regions used in this analysis, which have been defined based on the expected regional distribution of policy impacts, availability of economic and energy data, and computational limits on model size. These regions have been constructed from the underlying 10-region database designed to follow, as closely as possible, the electricity market regions defined by the North American Electric Reliability Council (NERC).¹⁷



Figure E-16. Regions Defined in EMPAX-CGE for the CAIR Analysis

¹⁷Economic data and information on nonelectricity energy markets are generally available only at the state level, which necessitates an approximation of the NERC regions that follows state boundaries. For the CAIR analysis, these approximations include Northeast = NPCC + MAAC, Southeast = SERC + FERC, Midwest = ECAR + MAIN, Plains = MAPP + SPP + ERCOT, and West = WSCC. See <a href="http://www.nerc.com/">http://www.nerc.com/</a> for further discussion of these regions.

### E.2.10 Production Functions

All productive markets are assumed to be perfectly competitive and have production technologies that exhibit constant returns to scale, except for the agriculture and natural resource extracting sectors, which have decreasing returns to scale because they use factors in fixed supply (land and fossil fuels, respectively). The electricity industry is separated into two distinct sectors: fossil-fuel generation and nonfossil generation. This allows tracking of variables such as heat rates for fossil-fired utilities (Btus of energy input per kilowatt hour of electricity output).

All markets must clear (i.e., supply must equal demand in every sector) in every period, and the income of each agent in the model must equal their factor endowments plus any net transfers. Along with the underlying data, the nesting structures shown in Figure E-15 and associated substitution elasticities define current production technologies and possible alternatives.

### E.2.11 Utility Functions

Each region in the dynamic version of EMPAX-CGE contains four representative households, classified by income, that maximize intertemporal utility over all time periods in the model subject to budget constraints, where the income groups are

- \$0 to \$14,999,
- \$15,000 to \$29,999,
- \$30,000 to \$49,999, and
- \$50,000 and above.

These representative households are endowed with factors of production including labor, capital, natural resources, and land inputs to agricultural production. Factor prices are equal to the marginal revenue received by firms from employing an additional unit of labor or capital. The value of factors owned by each representative household depends on factor use implied by production within each region. Income from sales of these productive factors is allocated to purchases of consumption goods to maximize welfare.

Within each time period, intratemporal utility received by a household is formed from consumption of goods and leisure. All consumption goods are combined using a Cobb-Douglas structure to form an aggregate consumption good. This composite good is then combined with leisure time to produce household utility. The elasticity of substitution

between consumption goods and leisure depends on empirical estimates of labor-supply elasticities and indicates how willing households are to trade off leisure time for consumption. Over time, households consider the discounted present value of utility received from all periods' consumption of goods and leisure.

Following standard conventions of CGE models, factors of production are assumed to be intersectorally mobile within regions, but migration of productive factors is not allowed across regions. This assumption is necessary to calculate welfare changes for the representative household located in each region in EMPAX-CGE. EMPAX-CGE also assumes that ownership of natural resources and capital embodied in nonfossil electricity generation is spread across the United States through capital markets.

# **E.2.12** Trade

In EMPAX-CGE, all goods and services are assumed to be composite, differentiated "Armington" goods made up of locally manufactured commodities and imported goods. Output of local industries is initially separated into output destined for local consumption by producers or households and output destined for export. This local output is then combined with goods from other regions in the United States using Armington-trade elasticities that indicate agents make relatively little distinction between output from firms located within their region and output from firms in other regions within the United States. Finally, the domestic composite goods are aggregated with imports from foreign sources using lower trade elasticities to capture the fact that foreign imports are more differentiated from domestic output than are imports from other regional suppliers in the United States.

### E.2.13 Tax Rates and Distortions

Taxes and associated distortions in economic behavior have been included in EMPAX-CGE because theoretical and empirical literature found that taxes can substantially alter estimated policy costs. The IMPLAN economic database used by EMPAX-CGE includes information on taxes such as indirect business taxes (all sales and excise taxes) and Social Security taxes. However, IMPLAN reports factor payments for labor and capital at their gross-of-tax values, which necessitates use of additional data sources to determine personal income and capital tax rates. Information from the TAXSIM model at the National Bureau of Economic Research (Feenberg and Coutts, 1993), along with user-cost-of-capital calculations from Fullerton and Rogers (1993), are used to establish tax rates.

Along with these rates, distortions associated with taxes are a function of labor supply decisions of households. As with other CGE models focused on interactions between tax and environmental policies (e.g., Bovenberg and Goulder [1996]; Goulder and Williams [2003]), an important feature of EMPAX-CGE is its inclusion of a labor-leisure choice—how people decide between working and leisure time. Labor supply elasticities related to this choice determine, to a large extent, how distortionary taxes are in a CGE model. Elasticities based on the relevant literature have been included in EMPAX-CGE (i.e., 0.4 for the compensated labor supply elasticity and 0.15 for the uncompensated labor supply elasticity). These elasticity values give an overall marginal excess burden associated with the existing tax structure of approximately 0.3.

## E.2.14 Intertemporal Dynamics and Economic Growth

There are four sources of economic growth in EMPAX-CGE: technological change from improvements in energy efficiency, growth in the available labor supply (from both population growth and changes in labor productivity), increases in stocks of natural resources, and capital accumulation. Energy consumption per unit of output tends to decline over time because of improvements in production technologies and energy conservation. These changes in energy use per unit of output are modeled as AEEIs, which are used to replicate energy consumption forecasts by industry and fuel from EIA.¹⁸ The AEEI values provide the means for matching expected trends in energy needed to produce a given quantity of output by incorporating improvements in energy efficiency and conservation. Labor force and regional economic growth, electricity generation, changes in available natural resources, and resource prices are also based on the AEO forecasts.

Savings provide the basis for capital formation and are motivated through people's expectations about future needs for capital. Savings and investment decisions made by households determine aggregate capital stocks in EMPAX-CGE. The IMPLAN dataset provides details on the types of goods and services used to produce the investment goods underlying each region's capital stocks. Adjustment dynamics associated with formation of capital are controlled by using quadratic adjustment costs experienced when installing new capital, which imply that real costs are experienced to build and install new capital equipment.

¹⁸See Babiker et al. (2001) for a discussion of how this methodology was used in the EPPA model (EPPA assumes that AEEI parameters are the same across all industries in a country, while AEEI values in EMPAX-CGE are industry specific).

Prior to investigating policy scenarios, it is necessary to establish a baseline path for the economy that incorporates economic growth and technology changes that are expected to occur in the absence of the policy actions. Beginning from the initial balanced SAM dataset, a "steady-state" growth path is first specified for the economy to ensure that the model remains in equilibrium in future years.¹⁹ Once the model is able to replicate a steady-state growth path, the assumption of a constant growth rate is replaced by actual forecasts from AEO. After incorporating these forecasts, EMPAX-CGE is solved to generate a baseline consistent with them through 2025. Once this baseline is established, it is possible to run "counterfactual" policy experiments.

### E.2.15 Alternative IPM-to-EMPAX Linkages

As discussed in Section E.2.2, EMPAX-CGE is capable of incorporating a variety of results from IPM, depending on the desired type of linkage between the two models. This section presents macroeconomic impacts of CAIR, as shown by changes in GDP, from two alternative methods of using IPM results. These findings are contrasted to the "Central Case" (i.e., the results presented above) to demonstrate how the methodology used to link the two models can influence results. One alternative, referred to as the "IPM Price & Fuel Case," places a higher degree of reliance on IPM results than the "Central Case." In the other alternative, referred to as the "Unconstrained Case," EMPAX-CGE is allowed to determine more market outcomes than in the "Central Case." These scenarios provide a range of results that illustrate the macroeconomic implications of different methods for linking macroeconomic models with the IPM results.

Specifically, the three alternative approaches are as follows:

• "Central Case"—This case from the main analysis incorporates IPM estimates of resource costs (capital costs, and fixed and variable operating costs), along with percentage changes in coal use (expressed in Btus), in EMPAX-CGE. The IPM resource costs are used to adjust electricity generation costs within EMPAX-CGE by requiring additional purchases of capital, labor, and material inputs. Natural gas expenditures are also adjusted based on IPM findings by requiring additional purchases.

¹⁹A steady-state growth path requires all variables in the model to grow at a constant rate over time, including labor, output, inputs to production, and consumption. If the model has been properly specified, the steady-state replication check will show that the economy remains in equilibrium in each year along this path.

- *"Unconstrained Case"*—This case incorporates IPM estimates of both resource costs and fuel expenditures for coal and gas into EMPAX-CGE. Unlike the "Central Case," this case allows changes in the quantity of coal use in the electricity sector to be estimated by EMPAX-CGE, once declines in the dollar value of coal purchases from the IPM model have been incorporated into the model. These declines in coal purchases are integrated using the same methodology applied to the capital, labor, and material inputs needed to generate electricity.
- *"IPM Price & Fuel Case"*—This case places the most reliance on IPM findings. Rather than allowing EMPAX-CGE to determine electricity price outcomes based on IPM resource costs, it replicates the IPM price results in EMPAX-CGE and concentrates on examining their implications for the rest of the economy.²⁰ Similarly, this case uses IPM data on changes in coal and gas use (in physical units) by electricity sector instead of allowing the CGE model to make these decisions. Market prices for coal and gas are still determined by EMPAX-CGE. This case takes into consideration the fact that, although most resource costs of electricity policies are borne by coal-fired generation, electricity prices are typically determined by the marginal unit in operation. Because of this, there may not be a direct correlation between policy costs and implications for electricity prices, although the economy outside of the electricity industry will respond to both electricity prices and any effects from drawing additional resources into electricity production.

Figure E-17 illustrates the implications of these alternative linkages between IPM and EMPAX-CGE for estimates of CAIR GDP effects. The "Central Case" from the main analysis and "Unconstrained Case" follow similar paths; however, the "Unconstrained Case" is uniformly less expensive. It provides more degrees of freedom to adjust coal consumption by utilities in response to demand changes estimated by EMPAX-CGE and also has added flexibility to shift among production inputs. This results in GDP impacts to around 20 percent lower than in the "Central Case."

²⁰IPM's estimated wholesale prices are converted into changes in retail prices for EMPAX-CGE using EIA estimates of transmission and distribution costs of approximately \$27 per megawatt hour.

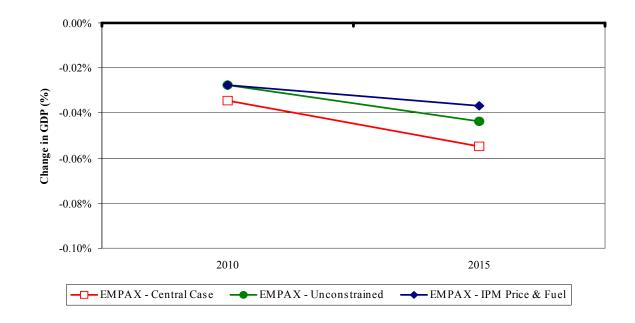


Figure E-17. GDP Impacts of Alternative Linkages (in %)

The "IPM Price & Fuel Case" shows changes in GDP that follow the same pattern as the "Central Case," but are generally lower. The methodologies of these two cases are substantially different: many of the effects in the "IPM Price & Fuel Case" are driven by IPM's estimated changes in electricity prices (and to a lesser degree by impacts on gas prices from increased demand by generators), while in the "Central Case" electricity prices predicted by EMPAX-CGE are controlled by how many additional resource costs are entering electricity production. IPM results show moderate increases in electricity prices in 2010 and 2015, leading to smaller GDP effects in the "IPM Price & Fuel Case" than in the "Central Case."

### E.3 References

- Babiker, M.H., and T.F. Rutherford. 1997. "Input-Output and General Equilibrium Estimates of Embodied CO2: A Data Set and Static Framework for Assessment." University of Colorado at Boulder, Working Paper 97-2. Available at http://debreu.colorado.edu/papers/gtaptext.html.
- Babiker, M.H., J.M. Reilly, M. Mayer, R.S. Eckaus, I.S. Wing, and R.C. Hyman. 2001.
  "The MIT Emissions Prediction and CO2 Policy Analysis (EPPA) Model: Revisions, Sensitivities, and Comparisons of Results." MIT Joint Program on the Science and Policy of Global Change, Report No. 71. Available at http://web.mit.edu/globalchange/www/eppa.html.
- Bovenberg, L.A., and L.H. Goulder. 1996. "Optimal Environmental Taxation in the Presence of Other Taxes: General-Equilibrium Analysis." *American Economic Review* 86(4):985-1000.
- Brooke, A., D. Kendrick, A. Meeraus, and R. Raman. 1998. GAMS: A User's Guide. GAMS Development Corporation. Available at http://www.gams.com.
- Feenberg, D., and E. Coutts. 1993. "An Introduction to the TAXSIM Model." Journal of Policy Analysis and Management 12(1):189-194. Available at http://www.nber.org/~taxsim/.
- Fullerton, D., and D. Rogers. 1993. "Who Bears the Lifetime Tax Burden?" Washington, DC: The Brookings Institute. Available at http://bookstore.brookings.edu/ book_details.asp?product%5Fid=10403.
- Goulder, L.H., and R.C. Williams. 2003. "The Substantial Bias from Ignoring General Equilibrium Effects in Estimating Excess Burden, and a Practical Solution." *Journal* of Political Economy 111:898-927.
- Minnesota IMPLAN Group. 2003. *State-Level Data for 2000*. Available from http://www.implan.com/index.html.
- Nestor, D.V., and C.A. Pasurka. 1995. The U.S. Environmental Protection Industry: A Proposed Framework for Assessment. U.S. Environmental Protection Agency, Office of Policy, Planning, and Evaluation. EPA 230-R-95-001. Available at http://yosemite.epa.gov/ee/epa/eermfile.nsf/11f680ff78df42f585256b45007e6235/41 b8b642ab9371df852564500004b543/\$FILE/EE-0217A-1.pdf.

- Rutherford, T.F. 1999. "Applied General Equilibrium Modeling with MPSGE as a GAMS Subsystem: An Overview of the Modeling Framework and Syntax." Computational Economics 14(1):1-46. Also available at http://www.gams.com/solvers/mpsge/syntax.htm.
- Rutherford, T.F., and S.V. Paltsev. 2000. "GTAP-Energy in GAMS: The Dataset and Static Model." University of Colorado at Boulder, Working Paper 00-2. Available at http://debreu.colorado.edu/papers/gtaptext.html.
- U.S. Department of Energy, Energy Information Administration. *State Energy Data Report*. Washington DC. Available at http://www.eia.doe.gov/emeu/states/ _use_multistate.html.
- U.S. Department of Energy, Energy Information Administration. *State Energy Price and Expenditure Report*. Washington DC. Available at http://www.eia.doe.gov/emeu/states/price_multistate.html.
- U.S. Department of Energy, Energy Information Administration. 2001. *Manufacturing Energy Consumption Survey 1998*. Washington DC. Available at http://www.eia.doe.gov/emeu/mecs/.
- U.S. Department of Energy, Energy Information Administration. January 2003. *Annual Energy Outlook 2003*. DOE/EIA-0383(2003). Washington DC. Available at http://www.eia.doe.gov/oiaf/archive/aeo03/pdf/0383(2003).pdf.
- U.S. Department of Energy, Energy Information Administration. January 2004. *Annual Energy Outlook 2004*. DOE/EIA-0383(2003). Washington, DC. Available at http://www.EOA.DOE.GOV/OIAF/AOE/PDF/0383 (2001).

#### **APPENDIX F**

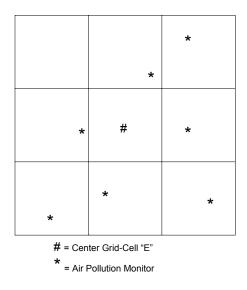
# ADDITIONAL TECHNICAL INFORMATION SUPPORTING THE BENEFITS ANALYSIS

This appendix provides additional technical details about several important elements of the benefits analysis, including the spatial interpolation method and health effect pooling methods. Additional details on benefits methods can be found in the BenMAP User's Manual, available in the docket and online at http://www.epa.gov/ttn/ecas/benmodels.html.

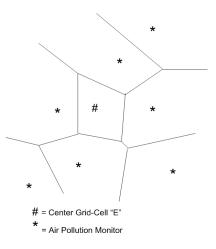
# F.1 Voronoi Neighbor Averaging

In calculating the base year concentrations of PM species and ozone at model grid cells prior to scaling with model outputs, we used a spatial interpolation method known as Voronoi Neighbor Averaging (VNA).

The first step in VNA is to identify the set of neighboring monitors for each of the grid cells in the continental United States. The figure below presents nine grid cells and seven monitors, with the focus on identifying the set of neighboring monitors for grid cell E.



In particular, BenMAP identifies the nearest monitors, or "neighbors," by drawing a polygon, or Voronoi cell, around the center of each grid cell. The polygons have the special property that the boundaries are the same distance from the two closest points.

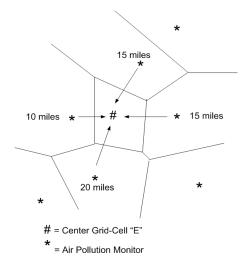


We then chose those monitors that share a boundary with the center of grid cell E. These are the nearest neighbors, and we used these monitors to estimate the air pollution level for this grid cell.

To estimate the air pollution level in each grid cell, BenMAP calculates the air pollution metrics for each of the neighboring monitors and then calculates an inverse-distance weighted average of the metrics. The further the monitor is from the grid cell center, the smaller the weight.

The weight for the monitor 20 kilometers from the center of grid cell E is calculated as follows:

$$d_{i,1} = \frac{\frac{1}{20}}{\left(\frac{1}{20} + \frac{1}{16} + \frac{1}{14}\right)} = 0.27$$



The weights for the other monitors would be calculated in a similar fashion.

#### F.2 The Random/Fixed Effect Pooling Procedure

Often more than one study has estimated a C-R function for a given pollutant-health endpoint combination. Each study provides an estimate of the pollutant coefficient,  $\beta$ , in the C-R function, along with a measure of the uncertainty of the estimate. Because uncertainty decreases as sample size increases, combining data sets is expected to yield more reliable estimates of  $\beta$  and therefore more reliable estimates of the incidence change predicted using  $\beta$ . Combining data from several comparable studies to analyze them together is often referred to as meta-analysis.

For a number of reasons, including data confidentiality, it is often impractical or impossible to combine the original data sets. Combining the *results* of studies to produce better estimates of  $\beta$  provides a second-best but still valuable way to synthesize information (DerSimonian and Laird, 1986). This is referred to as pooling. Pooling  $\beta$ s requires that all of the studies contributing estimates of  $\beta$  use the same functional form for the C-R function. That is, the  $\beta$ s must be measuring the same thing.

It is also possible to pool the study-specific estimates of incidence change derived from the C-R functions, instead of pooling the underlying  $\beta$ s themselves. For a variety of reasons, this is often possible when it is not feasible to pool the underlying  $\beta$ s. For example,

different estimates of a coefficient in the same C-R function but are instead estimates of coefficients in different C-R functions. We can, however, calculate the incidence change predicted by each C-R function (for a given change in pollutant concentration and, for the log-linear function, a given baseline incidence rate) and pool these incidence changes. BenMAP allows the pooling of incidence changes predicted by several studies for the same pollutant-health endpoint group combination. It also allows the pooling of the corresponding study-specific estimates of monetary benefits. As with estimates based on only a single study, BenMAP allows you to characterize the uncertainty surrounding pooled estimates of incidence change and/or monetary benefit. To do this, BenMAP pools the study-specific distributions of incidence changes (or monetary benefit) to derive a pooled distribution. This pooled distribution incorporates information

from all the studies used in the pooling procedure.

## F.2.1 Weights Used for Pooling

The relative contribution of any one study in the pooling process depends on the weight assigned to that study. A key component of the pooling process, then, is the determination of the weight given to each study. Various methods can be used to assign weights to studies. Below we discuss the possible weighting schemes that are available in BenMAP.

if one study is log-linear and another is linear, we could not pool the  $\beta$ s because they are not

## Subjective (User-specified) Weights

BenMAP allows the user the option of specifying the weights to be used. Suppose, for example, the user wants to simply average all study-specific results. He would then assign a weight of 1/N to each of the N study-specific distributions that are to be pooled. Note that subjective weights are limited to two decimal places and are normalized if they do not sum to one.

## Automatically Generated Weights

A simple average has the advantage of simplicity but the disadvantage of not taking into account the uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty. A common method for weighting estimates involves using their variances. Variance takes into account both the consistency of data and the sample size used to obtain the estimate, two key factors that influence the reliability of results. BenMAP has two methods of automatically generating pooling weights using the variances of the input distributions—fixed effects pooling and random/fixed effects pooling.

The discussion of these two weighting schemes is first presented in terms of pooling the pollutant coefficients (the  $\beta$ s), because that most closely matches the discussion of the method for pooling study results as it was originally presented by DerSimonian and Laird (1986). We then give an overview of the analogous weighting process used within BenMAP to generate weights for incidence changes rather than  $\beta$ s.

# F.3 Fixed Effects Weights

The fixed effects model assumes that there is a single true C-R relationship and therefore a single true value for the parameter  $\beta$  that applies everywhere. Differences among  $\beta$ s reported by different studies are therefore simply the result of sampling error. That is, each reported  $\beta$  is an estimate of the *same underlying parameter*. The certainty of an estimate is reflected in its variance (the larger the variance, the less certain the estimate). Fixed effects pooling therefore weights each estimate under consideration in proportion to the *inverse* of its variance.

Suppose there are n studies, with the  $i^{th}$  study providing an estimate  $\beta_i$  with variance  $v_i~(I=1,\,...,\,n).~$  Let

$$S = \sum \frac{1}{v_i},$$

denote the sum of the inverse variances. Then the weight,  $w_i$ , given to the ith estimate,  $\beta_i$ , is

$$w_i = \frac{1/v_i}{S}$$

This means that estimates with small variances (i.e., estimates with relatively little uncertainty surrounding them) receive large weights and those with large variances receive small weights.

The estimate produced by pooling based on a fixed effects model, then, is just a weighted average of the estimates from the studies being considered, with the weights as defined above. That is,

$$\beta_{fe} = \sum w_i * \beta_i$$

The variance associated with this pooled estimate is the inverse of the sum of the inverse variances:

$$v_{fe} = \frac{1}{\Sigma \ 1/v_i}$$

Table F-1 shows the relevant calculations for this pooling for three sample studies.

 Table F-1. Example of Fixed Effects Model Calculations

Study	β _i	Vi	1/v _i	Wi	$w_i^*\beta_i$
1	0.75	0.1225	8.16	0.016	0.012
2	1.25	0.0025	400	0.787	0.984
3	1.00	0.0100	100	0.197	0.197
Sum			$\Sigma = 508.16$	$\Sigma = 1.000$	$\Sigma = 1.193$

The sum of weighted contributions in the last column is the pooled estimate of  $\beta$  based on the fixed effects model. This estimate (1.193) is considerably closer to the estimate from study 2 (1.25) than is the estimate (1.0) that simply averages the study estimates. This reflects the fact that the estimate from study 2 has a much smaller variance than the estimates from the other two studies and is therefore more heavily weighted in the pooling.

The variance of the pooled estimate,  $v_{fe}$ , is the inverse of the sum of the variances, or 0.00197. (The sums of the  $\beta_i$  and  $v_i$  are not shown, because they are of no importance. The sum of the  $1/v_i$  is S, used to calculate the weights. The sum of the weights,  $w_i$ , i=1, ..., n, is 1.0, as expected).

# F.4 Random/Fixed Effects Weights

An alternative to the fixed effects model is the random effects model, which allows the possibility that the estimates  $\beta_i$  from the different studies may in fact be estimates of *different* parameters, rather than just different estimates of a single underlying parameter. In studies of the effects of PM₁₀ on mortality, for example, if the composition of PM₁₀ varies among study locations the underlying relationship between mortality and PM₁₀ may be different from one study location to another. For example, fine particles make up a greater fraction of PM₁₀ in Philadelphia than in El Paso. If fine particles are disproportionately responsible for mortality relative to coarse particles, then one would expect the true value of  $\beta$  in Philadelphia to be greater than the true value of  $\beta$  in El Paso. This would violate the assumption of the fixed effects model.

The following procedure can test whether it is appropriate to base the pooling on the random effects model (vs. the fixed effects model). A test statistic,  $Q_w$ , the weighted sum of squared differences of the separate study estimates from the pooled estimate based on the fixed effects model, is calculated as

$$Q_w = \sum_i \frac{1}{v_i} (\beta_{fe} - \beta_i)^2$$

Under the null hypothesis that there is a single underlying parameter,  $\beta$ , of which all the  $\beta_i$ s are estimates,  $Q_w$  has a chi-squared distribution with n–1 degrees of freedom. (Recall that n is the number of studies in the meta-analysis.) If  $Q_w$  is greater than the critical value corresponding to the desired confidence level, the null hypothesis is rejected. That is, in this case the evidence does not support the fixed effects model, and the random effects model is assumed, allowing the possibility that each study is estimating a different  $\beta$ . (BenMAP uses a 5 percent one-tailed test.)

The weights used in a pooling based on the random effects model must take into account not only the within-study variances (used in a meta-analysis based on the fixed effects model) but the between-study variances as well. These weights are calculated as follows:

Using  $Q_w$ , the between-study variance,  $\eta^2$ , is

$$\eta^{2} = \frac{Q_{w} - (n-1)}{\sum 1/v_{i} - \frac{\sum 1/v_{i}^{2}}{\sum 1/v_{i}}}$$

It can be shown that the denominator is always positive. Therefore, if the numerator is negative (i.e., if  $Q_w < n-1$ ), then  $\eta^2$  is a negative number, and it is not possible to calculate a random effects estimate. In this case, however, the small value of  $Q_w$  would presumably have led to accepting the null hypothesis described above, and the meta-analysis would be based on the fixed effects model. The remaining discussion therefore assumes that  $\eta^2$  is positive.

Given a value for  $\eta^2$ , the random effects estimate is calculated in almost the same way as the fixed effects estimate. However, the weights now incorporate both the within-study variance ( $v_i$ ) and the between-study variance ( $\eta^2$ ). Whereas the weights implied by the fixed effects model used only  $v_i$ , the within-study variance, the weights implied by the random effects model use  $v_i + \eta^2$ .

Let  $v_i^* = v_i + \eta^2$ . Then

$$S^* = \sum_{i=1}^{\infty} \frac{1}{v_i^*}$$
,

and

$$w_i^* = \frac{1/v_i^*}{S^*}$$

The estimate produced by pooling based on the random effects model, then, is just a weighted average of the estimates from the studies being considered, with the weights as defined above. That is,

$$\beta_{rand} = \sum w_i^* * \beta_i$$

The variance associated with this random effects pooled estimate is, as it was for the fixed effects pooled estimate, the inverse of the sum of the inverse variances:

$$v_{rand} = \frac{1}{\Sigma 1/v_i^*}$$

The weighting scheme used in a pooling based on the random effects model is basically the same as that used if a fixed effects model is assumed, but the variances used in the calculations are different. This is because a fixed effects model assumes that the variability among the estimates from different studies is due only to sampling error (i.e., each study is thought of as representing just another sample from the same underlying population), while the random effects model assumes that there is not only sampling error associated with each study, but that there is also *between-study* variability—each study is estimating a different underlying  $\beta$ . Therefore, the sum of the within-study variance and the between-study variance yields an overall variance estimate.

# F.5 Fixed Effects and Random/Fixed Effects Weighting to Pool Incidence Change Distributions and Dollar Benefit Distributions

Weights can be derived for pooling incidence changes predicted by different studies, using either the fixed effects or the fixed/random effects model, in a way that is analogous to the derivation of weights for pooling the  $\beta$ s in the C-R functions. As described above, BenMAP generates a Latin hypercube representation of the distribution of incidence change corresponding to each C-R function selected. The means of those study-specific Latin hypercube distributions of incidence change are used in exactly the same way as the reported  $\beta$ s are used to calculate fixed effects and random effects weights described above. The variances of incidence change are used in the same way as the variances of the  $\beta$ s. The formulas above for calculating fixed effects weights, for testing the fixed effects hypothesis, and for calculating random effects weights can all be used by substituting the mean incidence

change for the ith C-R function for  $\beta_i$  and the variance of incidence change for the ith C-R function for  $v_{i}$ .¹

Similarly, weights can be derived for dollar benefit distributions. As described above, BenMAP generates a Latin hypercube representation of the distribution of dollar benefits. The means of those Latin hypercube distributions are used in exactly the same way as the reported  $\beta$ s are used to calculate the fixed effects and random effects weights described above. The variances of dollar benefits are used in the same way as the variances of the  $\beta$ s. The formulas above for calculating fixed effects weights, for testing the fixed effects hypothesis, and for calculating random effects weights can all be used by substituting the mean dollar benefit change for the ith valuation for  $\beta_i$  and the variance of dollar benefits for the ith valuation for  $v_i$ .

BenMAP always derives fixed effects and random/fixed effects weights using nationally aggregated results, and uses those weights for pooling at each grid cell (or county, etc., if the user chooses to aggregate results prior to pooling). This is done because BenMAP does not include any regionally based uncertainty—that is, all uncertainty is at the national level in BenMAP, and all regional differences (e.g., population) are treated as certain.

#### F.6 Reference

DerSimonian, R. and N. Laird, "Meta-analysis in Clinical Trials," Controlled Clinical Trials 7:3 (1986), 177-188.

¹ There may be a problem with transferring the fixed effects hypothesis test to "incidence change space." The test statistic to test the fixed effects model is a chi-squared random variable. In the original paper on this pooling method, DerSimonian and Laird (1986) were discussing the pooling of estimates of parameters, which are generally normally distributed. The incidence changes predicted from a C-R function will not be normally distributed if the C-R function is not a linear function of the pollutant coefficient, which, in most cases it is not. (Most C-R functions are log-linear.) In that case, the test statistic may not be chi-square distributed. However, most log-linear C-R functions are *nearly* linear because their coefficients are very small. In that case the test statistic is likely to be *nearly* chi-square distributed.

#### **APPENDIX G**

# HEALTH-BASED COST-EFFECTIVENESS OF REDUCTIONS IN AMBIENT PM_{2.5} ASSOCIATED WITH CAIR

# G.1 Summary

Health-based cost-effectiveness analysis (CEA) and cost-utility analysis (CUA) have been used to analyze numerous health interventions but have not been widely adopted as tools to analyze environmental policies. The Office of Management and Budget (OMB) recently issued Circular A-4 guidance on regulatory analyses, requiring federal agencies to "prepare a CEA for all major rulemakings for which the primary benefits are improved public health and safety to the extent that a valid effectiveness measure can be developed to represent expected health and safety outcomes." Environmental quality improvements may have multiple health and ecological benefits, making application of CEA more difficult and less straightforward. For CAIR CEA may provide a useful framework for evaluation: nonhealth benefits are substantial, but the majority of quantified benefits come from health effects. Therefore, EPA is including in the CAIR RIA a preliminary and experimental application of one type of CEA—a modified quality-adjusted life-years (QALYs) approach. In this instance, the direct usefulness of cost-effectiveness analysis is mitigated by the lack of rule alternatives to compare relative effectiveness, but one can still make some comparisons to other benchmarks bearing in mind methodological differences.

QALYs were developed to evaluate the effectiveness of individual medical treatments, and EPA is still evaluating the appropriate methods for CEA for environmental regulations. Agency concerns with the standard QALY methodology include the treatment of people with fewer years to live (the elderly); fairness to people with preexisting conditions that may lead to reduced life expectancy and reduced quality of life; and how the analysis should best account for nonhealth benefits, such as improved visibility.

The Institute of Medicine (a member institution of the National Academies of Science) has established the Committee to Evaluate Measures of Health Benefits for Environmental, Health, and Safety Regulation to assess the scientific validity, ethical implications, and practical utility of a wide range of effectiveness measures used or proposed in CEA. This committee is expected to produce a report by the end of 2005. In the interim, however, agencies are expected to provide CEAs for rules covered by Circular A-4 requirements.

The methodology presented in this appendix is not intended to stand as precedent either for future air pollution regulations or for other EPA regulations where it may be inappropriate. It is intended solely to demonstrate one particular approach to estimating the cost-effectiveness of reductions in ambient  $PM_{2.5}$  in achieving improvements in public health. Reductions in ambient  $PM_{2.5}$  likely will have other health and environmental benefits that will not be reflected in this CEA. Other EPA regulations affecting other aspects of environmental quality and public health may require additional data and models that may preclude the development of similar health-based CEAs. A number of additional methodological issues must be considered when conducting CEAs for environmental policies, including treatment of nonhealth effects, aggregation of acute and long-term health impacts, and aggregation of life extensions and quality-of-life improvements in different populations. The appropriateness of health-based CEA should be evaluated on a case-bycase basis subject to the availability of appropriate data and models, among other factors.

CAIR is expected to result in substantial reductions in potential population exposure to ambient concentrations of PM. The benefit-cost analysis presented in Chapter 4 shows that CAIR achieves substantial health benefits whose monetized value far exceeds costs (net benefits are over \$100 billion in 2015). Despite the risk of oversimplifying benefits, cautiously-interpreted cost-effectiveness calculations may provide further evidence of whether the costs associated with CAIR are a reasonable health investment for the nation.

This analysis provides estimates of commonly used health-based effectiveness measures, including lives saved, life years saved (from reductions in mortality risk), and QALYs saved (from reductions in morbidity risk) associated with the reduction of ambient PM_{2.5} due to CAIR. In addition, a new aggregate effectiveness metric, Morbidity Inclusive Life Years (MILY) is introduced to address some of the concerns about aggregation of life extension and quality-of-life impacts.¹ It represents the sum of life years gained due to reductions in premature mortality and the QALY gained due to reductions in chronic morbidity. This measure may be preferred to existing QALY aggregation approaches because it does not devalue life extensions in individuals with preexisting illnesses that reduce quality of life. However, the MILY measure is still based on life years and thus still

¹This metric is also referred to as a "fair QALY" in Hubbell (2004b). The metric is also being considered by the Institute of Medicine Committee to Evaluate Measures of Health Benefits for Environmental, Health, and Safety Regulation under the term "fair QALY."

inherently gives more weight to interventions that reduce mortality and morbidity impacts for younger populations with higher remaining life expectancy. This analysis focuses on life extensions and improvements in quality of life through reductions in two diseases with chronic impacts: chronic bronchitis (CB) and nonfatal acute myocardial infarctions. Monte Carlo simulations are used to propagate uncertainty in several analytical parameters and characterize the distribution of estimated impacts.

Presented in three different metrics, the analysis suggests the following:

- In 2010 CAIR will result in:
  - 13,000 (95% CI: 5,400 20,000) premature deaths avoided, or
  - 140,000 (95% CI: 104,000 184,000) life years gained (discounted at 3 percent), or
  - 190,000 (95% CI: 140,000 250,000) MILYs gained (discounted at 3 percent).
- In 2015, CAIR will result in:
  - 17,000 (95% CI: 7,200 26,000) premature deaths avoided, or
  - 190,000 (95% CI: 140,000 240,000) life years gained (discounted at 3 percent), or
  - 250,000 (95% CI: 180,000 330,000) MILYs gained (discounted at 3 percent).
- Using a 7 percent discount rate, mean discounted life years gained are 110,000 in 2010 and 140,000 in 2015; mean MILYs gained are 140,000 in 2010 and 180,000 in 2015. (The estimates of premature deaths avoided are not affected by the discount rate.)
- The associated reductions in CB and nonfatal acute myocardial infarctions will reduce medical costs by approximately \$2.1 billion in 2010 and \$2.7 billion in 2015 based on a 3 percent discount rate, or \$1.8 billion in 2010 and \$2.3 billion in 2015 based on a 7 percent discount rate.
- Other health and nonhealth benefits are valued at \$1.9 billion in 2010 and \$2.8 billion in 2015.

Direct private compliance costs of the CAIR rule are \$2.4 billion in 2010 and \$3.6 billion in 2015. Therefore, the net costs (costs minus avoided cost of illness minus other benefits) are negative, indicating that CAIR results in cost savings. As such, traditional cost-

effectiveness ratios are not informative. However, it is possible to calculate the maximum costs for the rule that would still result in cost-effective improvements in public health compared with standard benchmarks of \$50,000 and \$100,000 per QALY:

- Taking into account avoided medical costs and other benefits, annual costs of CAIR would need to exceed \$14 billion (95% CI: \$9.8 billion – \$18 billion) in 2010 and \$18 billion (95% CI: \$13 billion – \$23 billion) in 2015 to have a cost per MILY that exceeds a benchmark of \$50,000, based on a 3 percent discount rate.
- Annual costs of CAIR would need to exceed \$23 billion (95% CI: \$17 billion \$31 billion) in 2010 and \$30 billion (95% CI: \$22 billion \$40 billion) in 2015 to have a cost per MILY that exceeds a benchmark of \$100,000, based on a 3 percent discount rate.
- 3. Using a 7 percent discount rate, annual costs of CAIR would need to exceed \$11 billion in 2010 and \$14 billion in 2015 to have a cost per MILY that exceeds a benchmark of \$50,000, and would need to exceed \$18 billion in 2010 and \$23 billion in 2015 to have a cost per MILY that exceeds a benchmark of \$100,000.

Given costs of \$2.4 billion and \$3.6 billion in 2010 and 2015, respectively, CAIR is clearly a very cost-effective way to achieve improvements in public health.

## G.2 Introduction

Analyses of environmental regulations have typically used benefit-cost analysis to characterize impacts on social welfare. Benefit-cost analyses allow for aggregation of the benefits of reducing mortality risks with other monetized benefits of reducing air pollution, including acute and chronic morbidity, and nonhealth benefits such as improved visibility. One of the great advantages of the benefit-cost paradigm is that a wide range of quantifiable benefits can be compared to costs to evaluate the economic efficiency of particular actions. However, alternative paradigms such as CEA and CUA analyses may also provide useful insights. CEA involves estimation of the costs per unit of benefit (e.g., lives or life years saved). CUA is a special type of CEA using preference-based measures of effectiveness, such as QALYs.

CEA and CUA are most useful for comparing programs that have similar goals, for example, alternative medical interventions or treatments that can save a life or cure a disease.

They are less readily applicable to programs with multiple categories of benefits, such as those reducing ambient air pollution, because the cost-effectiveness calculation is based on the quantity of a single benefit category. In other words, we cannot readily convert improvements in nonhealth benefits such as visibility to a health metric such as life years saved. For these reasons, environmental economists prefer to present results in terms of monetary benefits and net benefits.

However, QALY-based CUA has been widely adopted within the health economics literature (Neumann, 2003; Gold et al., 1996) and in the analysis of public health interventions (US FDA, 2004). QALY-based analyses have not been as accepted in the environmental economics literature because of concerns about the theoretical consistency of QALYs with individual preferences (Hammitt, 2002), treatment of nonhuman health benefits, and a number of other factors (Freeman, Hammitt, and De Civita, 2002). For environmental regulations, benefit-cost analysis has been the preferred method of choosing among regulatory alternatives in terms of economic efficiency. Recently several academic analyses have proposed the use of life years-based benefit-cost or CEAs of air pollution regulations (Cohen, Hammitt, and Levy, 2003; Coyle et al., 2003; Rabl, 2003; Carrothers, Evans, and Graham, 2002). In addition, the World Health Organization has adopted the use of disability-adjusted life years, a variant on QALYs, to assess the global burden of disease due to different causes, including environmental pollution (Murray et al., 2002; de Hollander et al., 1999).

Recently, the U.S. OMB (Circular A-4, 2003) issued new guidance requiring federal agencies to provide both CEA and benefit-cost analyses for major regulations. The OMB Circular A-4 directs agencies to "prepare a CEA for all major rulemakings for which the primary benefits are improved public health and safety to the extent that a valid effectiveness measure can be developed to represent expected health and safety outcomes." We are including a CEA for CAIR to illustrate one potential approach for conducting a CEA. This is an experimental application, and EPA is still evaluating the appropriate methods for CEA for environmental regulations with multiple outcomes. The Institute of Medicine (a member institution of the National Academies of Science) has empaneled the Committee to Evaluate Measures of Health Benefits for Environmental, Health, and Safety Regulation to assess the scientific validity, ethical implications, and practical utility of a wide range of effectiveness measures used or proposed in CEA. This committee is expected to produce a report by the end of 2005. However, in the interim, are required to provide CEAs to comply with Circular A-4. As such, EPA has begun developing methodologies for estimating the cost-effectiveness of air pollution regulations. The methodology presented in this appendix is not

intended to stand as precedent either for future air pollution regulations or for other EPA regulations governing water, solid waste, or other regulatory objectives. It is intended solely to demonstrate one particular approach to estimating the effectiveness of reductions in ambient PM_{2.5} in achieving improvements in public health. This analysis focuses on effectiveness measured by improvements in life expectancy and reductions in the incidence of two diseases with chronic impacts on quality of life: CB and nonfatal acute myocardial infarctions. Other EPA regulations affecting other aspects of environmental quality and public health may require additional data and models that may preclude the development of similar QALY-based analyses. The appropriateness of QALY-based CEA should be evaluated on a case-by-case basis subject to the availability of appropriate data and models.

Preparation of a CEA requires identification of an appropriate measure of rule effectiveness. Given the significant impact of reductions in ambient PM_{2.5} on reductions in the risk of mortality, lives saved is an important measure of effectiveness. However, one of the ongoing controversies in health impact assessment regards whether reductions in mortality risk should be reported and valued in terms of statistical lives saved or in terms of statistical life years saved. Life years saved measures differentiate among premature mortalities based on the remaining life expectancy of affected individuals. In general, under the life years approach, older individuals will gain fewer life years than younger individuals for the same reduction in mortality risk during a given time period, making interventions that benefit older individuals. A further complication in the debate is whether to apply quality adjustments to life years lost. Under this approach, individuals with preexisting health conditions would have fewer QALYs lost relative to healthy individuals with poor health seem less beneficial to similar interventions affecting primarily healthy individuals.

In addition to substantial mortality risk reduction benefits, CAIR will also result in significant reductions in chronic and acute morbidity. Several approaches have been developed to incorporate both morbidity and mortality into a single effectiveness metric. The most common of these is the QALY approach, which expresses all morbidity and mortality impacts in terms of quality of life multiplied by the duration of time with that quality of life. The QALY approach has some appealing characteristics. For example, it can account for morbidity effects as well as losses in life expectancy without requiring the assignment of dollar values to calculate total benefits. By doing so it provides an alternative framework to benefit-cost analysis for aggregating quantitative measures of health impacts.

While used extensively in the economic evaluation of medical interventions (Gold et al., 1996), QALYs have not been widely used in evaluating environmental health regulations. A number of specific issues arise with the use of QALYs in evaluating environmental programs that affect a broad and heterogeneous population and that provide both health and nonhealth benefits. The U.S. Public Health Service report on cost-effectiveness in health and medicine notes the following:

For decisions that involve greater diversity in interventions and the people to whom they apply, cost-effectiveness ratios continue to provide essential information, but that information must, to a greater degree, be evaluated in light of circumstances and values that cannot be included in the analysis. Individuals in the population will differ widely in their health and disability before the intervention, or in age, wealth, or other characteristics, raising questions about how society values gains for the more and less health, for young and old, for rich and poor, and so on. The assumption that all QALYs are of equal value is less likely to be reasonable in this context. (Gold et al., 1996, p. 11)

Use of QALYs as a measure of effectiveness for environmental regulations is still developing, and while this analysis provides one framework for using QALYs to evaluate environmental regulations, there are clearly many issues, both scientific and ethical, that need to be addressed with additional research. The Institute of Medicine panel evaluating QALYs and other effectiveness measures will develop criteria for choosing among the measures that potentially are useful for regulatory impact analysis and will make recommendations regarding measures appropriate for assessing the health benefits of regulatory interventions and propose criteria for identifying regulations for which CEA is appropriate and informative.

This appendix presents cost-effectiveness methodologies for evaluating programs such as CAIR that are intended to reduce ambient PM_{2.5} starting from the standard QALY literature and seeking a parallel structure to benefit-cost analysis in the use of air quality and health inputs (see Hubbell [2004a] for a discussion of some of the issues that arise in comparing QALY and benefit-cost frameworks in analyzing air pollution impacts). For the purposes of this analysis, we calculate effectiveness using several different metrics, including lives prolonged, life years gained, and modified QALYs. For the life years and QALY-type approaches, we use life table methods to calculate the change in life expectancy expected to result from changes in mortality risk from PM. We use existing estimates of preferences for different health states to obtain QALY weights for morbidity endpoints associated with air

pollution. In general, consistent with the Gold et al. (1996) recommendations, we use weights obtained from a societal perspective when available. We explore several different sources for these weights to characterize some of the potential uncertainty in the QALY estimates. We follow many of the principles of the reference case analysis as defined in Gold et al. (1996), although in some cases we depart from the reference case approach when data limitations require us to do so (primarily in the selection of quality-of-life weights for morbidity endpoints). We also depart from the reference case in the method of combining life expectancy and quality-of-life gains.

Results in most tables are presented only at a discount rate of 3 percent, rather than at both 3 percent and 7 percent as recommended in EPA and OMB guidance. This is strictly for ease of presentation in EPA's first demonstration of this approach to cost-effectiveness. Aggregate results at 7 percent are presented in the summary, and the impact of using a 7 percent discount rate instead of 3 percent rate is summarized in a sensitivity analysis.

Monte Carlo simulation methods are used to propagate uncertainty in several of the model parameters throughout the analysis. We characterize overall uncertainty in the results with 95 percent confidence intervals based on the Monte Carlo simulations. In addition, we examine the impacts of changing key parameters, such as the discount rate, on the effectiveness measures and the cost-effectiveness metrics.

The remainder of this appendix provides an overview of the key issues involved in life year- and QALY-based approaches for evaluating the health impacts of air pollution regulations, provides detailed discussions of the steps required for each type of effectiveness calculation, and presents the CEA for CAIR. Section G.3 introduces the various effectiveness measures and discusses some of the assumptions required for each. Section G.4 details the methodology used to calculate changes in life years and quality adjustments for mortality and morbidity endpoints. Section G.5 provides the results for CAIR and discusses their implications for cost-effectiveness of CAIR.

#### G.3 Effectiveness Measures

Three major classes of benefits are associated with reductions in air pollution: mortality, morbidity, and nonhealth (welfare). For the purposes of benefit-cost analysis, EPA has presented mortality-related benefits using estimates of avoided premature mortalities, representing the cumulative result of reducing the risk of premature mortality from long-term exposure to  $PM_{2.5}$  for a large portion of the U.S. population. Morbidity benefits have been characterized by numbers of new incidences avoided for chronic diseases such as CB, avoided admissions for hospitalizations associated with acute and chronic conditions, and avoided days with symptoms for minor illnesses. Nonhealth benefits are characterized by the monetary value of reducing the impact (e.g., the dollar value of improvements in visibility at national parks).

For the purposes of CEA, we focus the effectiveness measure on the quantifiable health impacts of the reduction in  $PM_{2.5}$ . Treatment of nonhealth benefits is important and is discussed in some detail later in this section. If the main impact of interest is reductions in mortality risk from air pollution, the effectiveness measures are relatively straightforward to develop. Mortality impacts can be characterized similar to the benefits analysis, by counting the number of premature mortalities avoided, or can be characterized in terms of increases in life expectancy or life years.² Estimates of premature mortality have the benefit of being relatively simple to calculate, are consistent with the benefit-cost analysis, and do not impose additional assumptions on the degree of life shortening. However, some have argued that counts of premature mortalities avoided are problematic because a gain in life of only a few months would be considered equivalent to a gain of a many life years (Rabl, 2003; Miller and Hurley, 2003).

Calculations of changes in life years and life expectancy can be accomplished using standard life table methods (Miller and Hurley, 2003). However, the calculations require assumptions about the baseline mortality risks for each age cohort affected by air pollution. A general assumption may be that air pollution mortality risks affect the general mortality risk of the population in a proportional manner. However, some concerns have been raised that air pollution affects mainly those individuals with preexisting cardiovascular and respiratory disease, who may have reduced life expectancy relative to the general population. This issue is explored in more detail below.

Air pollution is also associated with a number of significant chronic and acute morbidity endpoints. Failure to consider these morbidity effects may understate the cost-

²Life expectancy is an *ex ante* concept, indicating the impact on an entire population's expectation of the number of life years they have remaining, before knowing which individuals will be affected. Life expectancy thus incorporates both the probability of an effect and the impact of the effect if realized. Life years is an *ex post* concept, indicating the impact on individuals who actually die from exposure to air pollution. Changes in population life expectancy will always be substantially smaller than changes in life years per premature mortality avoided, although the total life years gained in the population will be the same. This is because life expectancy gains average expected life years gained over the entire population, while life years gained measures life years gained only for those experiencing the life extension.

effectiveness of air pollution regulations or give too little weight to reductions in particular pollutants that have large morbidity impacts but no effect on life expectancy. The QALY approach explicitly incorporates morbidity impacts into measures of life years gained and is often used in health economics to assess the cost-effectiveness of medical spending programs (Gold et al., 1996). Using a QALY rating system, health quality ranges from 0 to 1, where 1 may represent full health, 0 death, and some number in between (e.g., 0.8) an impaired condition. QALYs thus measure morbidity as a reduction in quality of life over a period of life. QALYs assume that duration and quality of life are equivalent, so that 1 year spent in perfect health is equivalent to 2 years spent with quality of life half that of perfect health. QALYs can be used to evaluate environmental rules under certain circumstances, although some very strong assumptions (detailed below) are associated with QALYs. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommended using QALYs when evaluating medical and public health programs that primarily reduce both mortality and morbidity (Gold et al., 1996). Although there are significant nonhealth benefits associated with air pollution regulations, over 90 percent of quantifiable monetized benefits are health-related, as is the case with CAIR. Thus, it can be argued that QALYs are more applicable for these types of regulations than for other environmental policies. However, the value of nonhealth benefits should not be ignored. As discussed below, we have chosen to subtract the value of nonhealth benefits from the costs in the numerator of the cost-effectiveness ratio.

In the following sections, we lay out a phased approach to describing effectiveness. We begin by discussing how the life-extending benefits of air pollution reductions are calculated, and then we incorporate morbidity effects using the QALY approach. We also introduce an alternative aggregated health metric, Morbidity Inclusive Life Years (MILY) to address some of the ethical concerns about aggregating life extension impacts in populations with preexisting disabling conditions.

The use of QALYs is predicated on the assumptions embedded in the QALY analytical framework. As noted in the QALY literature, QALYs are consistent with the utility theory that underlies most of economics only if one imposes several restrictive assumptions, including independence between longevity and quality of life in the utility function, risk neutrality with respect to years of life (which implies that the utility function is linear), and constant proportionality in trade-offs between quality and quantity of life (Pliskin, Shepard, and Weinstein, 1980; Bleichrodt, Wakker, and Johannesson, 1996). To the extent that these assumptions do not represent actual preferences, the QALY approach will not provide results that are consistent with a benefit-cost analysis based on the KaldorHicks criterion.³ Even if the assumptions are reasonably consistent with reality, because QALYs represent an average valuation of health states rather than the sum of societal WTP, there are no guarantees that the option with the highest QALY per dollar of cost will satisfy the Kaldor-Hicks criterion (i.e., generate a potential Pareto improvement [Garber and Phelps, 1997]).

Benefit-cost analysis based on WTP is not without potentially troubling underlying structures as well, incorporating ability to pay (and thus the potential for equity concerns) and the notion of consumer sovereignty (which emphasizes wealth effects). Table G-1 compares the two approaches across a number of parameters. For the most part, WTP allows parameters to be determined empirically, while the QALY approach imposes some conditions *a priori*.

Parameter	QALY	WTP
Risk aversion	Risk neutral	Empirically determined
Relation of duration and quality	Independent	Empirically determined
Proportionality of duration/ quality trade-off	Constant	Variable
Treatment of time/age in utility function	Utility linear in time	Empirically determined
Preferences	Community/Individual	Individual
Source of preference data	Stated	Revealed and stated
Treatment of income and prices	Not explicitly considered	Constrains choices

#### Table G-1. Comparison of QALY and WTP Approaches

³The Kaldor-Hicks efficiency criterion requires that the "winners" in a particular case be potentially able to compensate the "losers" such that total societal welfare improves. In this case, it is sufficient that total benefits exceed total costs of the regulation. This is also known as a potential Pareto improvement, because gains could be allocated such that at least one person in society would be better off while no one would be worse off.

#### G.4 Changes in Premature Death, Life Years, and Quality of Life

To generate health outcomes, we used the same framework as for the benefit-cost analysis described in Chapter 4. For convenience, we summarize the basic methodologies here. For more details, see Chapter 4 and the BenMAP user's manual (http://www.epa.gov/ttn/ecas/benmodels.html).

BenMAP uses health impact functions to generate changes in the incidence of health effects. Health impact functions are derived from the epidemiology literature. A standard health impact function has four components: an effect estimate from a particular epidemiological study, a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics like CDC), the affected population, and the estimated change in the relevant PM summary measure.

A typical health impact function might look like this:

$$\Delta y = y_0 \cdot (e^{\beta \cdot \Delta x} - 1)$$

where  $y_0$  is the baseline incidence, equal to the baseline incidence rate times the potentially affected population;  $\beta$  is the effect estimate; and  $\Delta x$  is the estimated change in PM_{2.5}. There are other functional forms, but the basic elements remain the same.

#### G.4.1 Calculating Reductions in Premature Deaths

As in several recent air pollution health impact assessments (e.g., Kunzli et al., 2000; EPA, 2004), we focus on the prospective cohort long-term exposure studies in deriving the health impact function for the estimate of premature mortality. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time (Kunzli et al., 2001; NRC, 2002). We selected an effect estimate from the extended analysis of the ACS cohort (Pope et al., 2002). This latest re-analysis of the ACS cohort data provides additional refinements to the analysis of PM-related mortality by (a) extending the follow-up period for the ACS study subjects to 16 years, which triples the size of the mortality data set; (b) substantially increasing exposure data, including consideration for cohort exposure to PM_{2.5} following implementation of PM_{2.5} standard in 1999; (c) controlling for a variety of personal risk factors including occupational exposure and diet; and (d) using advanced statistical methods to evaluate specific issues that can adversely affect risk estimates, including the possibility of spatial autocorrelation of survival times in communities located near each other. The effect estimate from Pope et al. (2002) quantifies the relationship between annual mean PM_{2.5} levels and all-cause mortality in adults 30 and older. We

selected the effect estimate estimated using the measure of PM representing average exposure over the follow-up period, calculated as the average of 1979–1984 and 1999–2000  $PM_{2.5}$  levels. The effect estimate from this study is 0.0058, which is equivalent to a relative risk of 1.06 for a 10 µg change in  $PM_{2.5}$ . Although there are other cohort-based studies of the relationship between  $PM_{2.5}$  and mortality, none provide the same level of population and geographic coverage as the ACS study.

Age, cause, and county-specific mortality rates were obtained from CDC for the years 1996 through 1998. CDC maintains an online data repository of health statistics, CDC Wonder, accessible at http://wonder.cdc.gov/. The mortality rates provided are derived from U.S. death records and U.S. Census Bureau postcensal population estimates. Mortality rates were averaged across 3 years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group. For example, to estimate mortality rates for individuals ages 30 and up, we scaled the 25- to 34-year old death count and population by one-half and then generated a population-weighted mortality rate using data for the older age groups.

The reductions in incidence of premature mortality within each age group associated with the CAIR reductions in  $PM_{2.5}$  in 2010 and 2015 are summarized in Table G-2.

# G.4.2 Calculating Changes in Life Years from Direct Reductions in PM_{2.5}-Related Mortality Risk

To calculate changes in life years associated with a given change in air pollution, we used a life table approach coupled with age-specific estimates of reductions in premature mortality. We began with the complete unabridged life table for the United States in 2000, obtained from CDC (CDC, 2002). For each 1-year age interval (e.g., zero to one, one to two) the life table provides estimates of the baseline probability of dying during the interval, person years lived in the interval, and remaining life expectancy. From this unabridged life table, we constructed an abridged life table to match the age intervals for which we have predictions of changes in incidence of premature mortality. We used the abridgement method described in CDC (2002). Table G-3 presents the abridged life table for 10-year age intervals for adults over 30 (to match the Pope et al. [2002] study population). Note that the abridgement actually includes one 5-year interval, covering adults 30 to 34, with the remaining age intervals covering 10 years each. This is to provide conformity with the age intervals available for mortality rates.

	Reduction in All-Cause Premature Mortality (95% CI)			
Age Interval	2010	2015		
30 - 34	96 (33 – 160)	120 (42 – 200)		
35 - 44	370 (120 - 610)	420 (140 – 700)		
45 – 54	870 (300 – 1,400)	1,000 (340 – 1,700)		
55 - 64	1,700 (580 – 2,800)	2,300 (790 - 3,800)		
65 - 74	2,400 (830 - 4,000)	3,600 (1,200 – 6,000)		
75 – 84	3,400 (1,100 – 5,600)	4,100 (1,400 – 6,800)		
85+	3,800 (1,300 - 6,400)	5,100 (1,700 – 8,400)		
Total	12,700 (4,300 – 21,000)	17,000 (5,700 – 28,000)		

 Table G-2. Estimated Reduction in Incidence of All-cause Premature Mortality

 Associated with the Clean Air Interstate Rule

From the abridged life table (Table G-3), we obtained the remaining life expectancy for each age cohort, conditional on surviving to that age. This is then the number of life years lost for an individual in the general population dying during that age interval. This information can then be combined with the estimated number of premature deaths in each age interval calculated with BenMAP (see previous subsection). Total life years gained will then be the sum of life years gained in each age interval:

Total Life Years = 
$$\sum_{i=1}^{N} LE_i \times M_i$$
,

where  $LE_i$  is the remaining life expectancy for age interval *i*,  $M_i$  is the change in incidence of mortality in age interval *i*, and N is the number of age intervals.

-

		Probability of		Number	Person Years	<b>Total Number</b>	
		Dying	Number	Dying	Lived	of Person	Expectation
		Between Ages	Surviving to	Between Ages	Between Ages	Years Lived	of Life at
Age I	nterval	x to x+1	Age x	x to x+1	x to x+1	Above Age x	Age x
Start Age	End Age	q _x	I _x	d _x	L _x	T _x	e _x
30	35	0.00577	97,696	564	487,130	4,723,539	48.3
35	45	0.01979	97,132	1,922	962,882	4,236,409	43.6
45	55	0.04303	95,210	4,097	934,026	3,273,527	34.4
55	65	0.09858	91,113	8,982	872,003	2,339,501	25.7
65	75	0.21779	82,131	17,887	740,927	1,467,498	17.9
75	85	0.45584	64,244	29,285	505,278	726,571	11.3
85	95	0.79256	34,959	27,707	196,269	221,293	6.3
95	100	0.75441	7,252	5,471	20,388	25,024	3.5
100+		1.00000	1,781	1,781	4,636	4,636	2.6

Table G-3. Abridged Life Table for the Total Population, United States, 2000

For the purposes of determining cost-effectiveness, it is also necessary to consider the time-dependent nature of the gains in life years. Standard economic theory suggests that benefits occurring in future years should be discounted relative to benefits occurring in the present. OMB and EPA guidance suggest discount rates of three and seven percent. As noted earlier, we present gains in future life years discounted at 3 percent. Results based on 7 percent are included in the summary and the overall impact of a 7 percent rate is summarized in Table G-16. Selection of a 3 percent discount rate is also consistent with recommendations from the U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine (Gold et al., 1996).

Discounted total life years gained is calculated as follows:

Discounted 
$$LY = \int_0^{LE} e^{-rt} dt$$
,

where r is the discount rate, equal to 0.03 in this case, t indicates time, and LE is the life expectancy at the time when the premature death would have occurred. Life years are further discounted to account for the lag between the reduction in ambient  $PM_{2.5}$  and the reduction in mortality risk. We use the same 20-year segmented lag structure that is used in the benefit-cost analysis (see Chapter 4).

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The most complete estimate of the impacts of  $PM_{2.5}$  on life years is calculated using the Pope et al. (2002) C-R function relating all-cause mortality in adults 30 and over with ambient  $PM_{2.5}$  concentrations averaged over the periods 1979–1983 and 1999–2000. Use of all-cause mortality is appropriate if there are no differences in the life expectancy of individuals dying from air pollution-related causes and those dying from other causes. The argument that long-term exposure to  $PM_{2.5}$  may affect mainly individuals with serious preexisting illnesses is not supported by current empirical studies. For example, the Krewski et al. (2000) ACS reanalysis suggests that the mortality risk is no greater for those with preexisting illness at time of enrollment in the study. Life expectancy for the general population in fact includes individuals with serious chronic illness. Mortality rates for the general population then reflect prevalence of chronic disease, and as populations age the prevalence of chronic disease increases.

The only reason one might use a lower life expectancy is if the population at risk from air pollution was limited solely to those with preexisting disease. Also, note that the OMB Circular A-4 notes that "if QALYs are used to evaluate a lifesaving rule aimed at a population that happens to experience a high rate of disability (i.e., where the rule is not designed to affect the disability), the number of life years saved should not necessarily be diminished simply because the rule saves lives of people with life-shortening disabilities. Both analytic simplicity and fairness suggest that the estimate number of life years saved for the disabled population should be based on average life expectancy information for the relevant age cohorts." As such, use of a general population life expectancy is preferred over disability-specific life expectancies. Our primary life years calculations are thus consistent with the concept of not penalizing individuals with disabling chronic health conditions by assessing them reduced benefits of mortality risk reductions.

For this analysis, direct impacts on life expectancy are measured only through the estimated change in mortality risk based on the Pope et al. (2002) C-R function. The SAB-HES has advised against including additional gains in life expectancy due to reductions in incidence of chronic disease or nonfatal heart attacks (EPA-SAB-COUNCIL-ADV-04-002). Although reductions in these endpoints are likely to result in increased life expectancy, the HES has suggested that the cohort design and relatively long follow-up period in the Pope et al. study should capture any life-prolonging impacts associated with those endpoints. Impacts of CB and nonfatal heart attacks on quality of life will be captured separately in the QALY calculation as years lived with improved quality of life. The methods for calculating this benefit are discussed below.

### G.4.2.1 Should Life Years Gained Be Adjusted for Initial Health Status?

The methods outlined above provide estimates of the total number of life years gained in a population, regardless of the quality of those life years, or equivalently, assuming that all life years gained are in perfect health. In some CEAs (Cohen, Hammitt, and Levy, 2003; Coyle et al., 2003), analysts have adjusted the number of life years gained to reflect the fact that 1) the general public is not in perfect health and thus "healthy" life years are less than total life years gained and 2) those affected by air pollution may be in a worse health state than the general population and therefore will not gain as many "healthy" life years adjusted for quality, from an air pollution reduction. This adjustment, which converts life vears gained into OALYs, raises a number of serious ethical issues. Proponents of OALYs have promoted the nondiscriminatory nature of OALYs in evaluating improvements in quality of life (e.g., an improvement from a score of 0.2 to 0.4 is equivalent to an improvement from 0.8 to 1.0), so the starting health status does not affect the evaluation of interventions that improve quality of life. However, for life-extending interventions, the gains in OALY will be directly proportional to the baseline health state (e.g., an individual with a 30-year life expectancy and a starting health status of 0.5 will gain exactly half the OALYs of an individual with the same life expectancy and a starting health status of 1.0 for a similar life-extending intervention). This is troubling because it imposes an additional penalty for those already suffering from disabling conditions. Brock (2002) notes that "the problem of disability discrimination represents a deep and unresolved problem for resource prioritization."

OMB (2003) has recognized this issue in their Circular A-4 guidance, which includes the following statement:

When CEA is performed in specific rulemaking contexts, you should be prepared to make appropriate adjustments to ensure fair treatment of all segments of the population. Fairness is important in the choice and execution of effectiveness measures. For example, if QALYs are used to evaluate a lifesaving rule aimed at a population that happens to experience a high rate of disability (i.e., where the rule is not designed to affect the disability), the number of life years saved should not necessarily be diminished simply because the rule saves the lives of people with life-shortening disabilities. Both analytic simplicity and fairness suggest that the estimated number of life years saved for the disabled population should be based on average life expectancy information for the relevant age cohorts. More generally, when numeric adjustments are made for life expectancy or quality of life, analysts

should prefer use of population averages rather than information derived from subgroups dominated by a particular demographic or income group. (p. 13)

This suggests two adjustments to the standard QALY methodology: one adjusting the relevant life expectancy of the affected population, and the other affecting the baseline quality of life for the affected population.

In addition to the issue of fairness, potential measurement issues are specific to the air pollution context that might argue for caution in applying quality-of-life adjustments to life years gained due to air pollution reductions. A number of epidemiological and toxicological studies link exposure to air pollution with chronic diseases, such as CB and atherosclerosis (Abbey et al., 1995; Schwartz, 1993; Suwa et al., 2002). If these same individuals with chronic disease caused by exposure to air pollution are then at increased risk of premature death from air pollution, there is an important dimension of "double jeopardy" involved in determining the correct baseline for assessing QALYs lost to air pollution (see Singer et al. [1995] for a broader discussion of the double-jeopardy argument).

Analyses estimating mortality from acute exposures that ignore the effects of longterm exposure on morbidity may understate the health impacts of reducing air pollution. Individuals exposed to chronically elevated levels of air pollution may realize an increased risk of death and chronic disease throughout life. If at some age they contract heart (or some other chronic) disease as a result of the exposure to air pollution, they will from that point forward have both reduced life expectancy and reduced quality of life. The benefit to that individual from reducing lifetime exposure to air pollution would be the increase in life expectancy plus the increase in quality of life over the full period of increased life expectancy. If the QALY loss is determined based on the underlying chronic condition and life expectancy without regard to the fact that the person would never have been in that state without long-term exposure to elevated air pollution, then the person is placed in double jeopardy. In other words, air pollution has placed more people in the susceptible pool, but then we penalize those people in evaluating policies by treating their subsequent deaths as less valuable, adding insult to injury, and potentially downplaying the importance of life expectancy losses due to air pollution. If the risk of chronic disease and risk of death are considered together, then there is no conceptual problem with measuring QALYs, but this has not been the case in recent applications of QALYs to air pollution (Carrothers, Evans, and Graham, 2002; Coyle et al., 2003). The use of QALYs thus highlights the need for a better understanding of the relationship between chronic disease and long-term exposure and suggests that analyses need to consider morbidity and mortality jointly, rather than treating each as a separate endpoint (this is an issue for current benefit-cost approaches as well).

Because of the fairness and measurement concerns discussed above, for the purposes of this analysis, we do not reduce the number of life years gained to reflect any differences in underlying health status that might reduce quality of life in remaining years. Thus, we maintain the assumption that all direct gains in life years resulting from mortality risk reductions will be assigned a weight of 1.0. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that "since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY" (Gold et al., 1996). However, for the purposes of this analysis, we propose an alternative to the traditional aggregate QALY metric that keeps separate quality adjustments to life years gained to reflect the less than perfect health of the general population. Gains in quality of life will be addressed as they accrue because of reductions in the incidence of chronic diseases. This is an explicit equity choice in the treatment of issues associated with quality-of-life adjustments for increases in life expectancy that still capitalizes on the ability of QALYs to capture both morbidity and mortality impacts in a single effectiveness measure.

# G.5 Calculating Changes in the Quality of Life Years (Morbidity)

In addition to directly measuring the quantity of life gained, measured by life years, it may also be informative to measure gains in the quality of life. Reducing air pollution also leads to reductions in serious illnesses that affect quality of life. These include CB and cardiovascular disease, for which we are able to quantify changes in the incidence of nonfatal heart attacks. To capture these important benefits in the measure of effectiveness, they must first be converted into a life-year equivalent so that they can be combined with the direct gains in life expectancy.

For this analysis, we developed estimates of the QALYs gained from reductions in the incidence of CB and nonfatal heart attacks associated with reductions in ambient  $PM_{2.5}$ . In general, QALY calculations require four elements:

- 1. the estimated change in incidence of the health condition,
- 2. the duration of the health condition,
- 3. the quality-of-life weight with the health condition, and

4. the quality-of-life weight without the health condition (i.e., the baseline health state).

The first element is derived using the health impact function approach. The second element is based on the medical literature for each health condition. The third and fourth elements are derived from the medical cost-effectiveness and cost-utility literature. In the following two subsections, we discuss the choices of elements for CB and nonfatal heart attacks.

The preferred source of quality-of-life weights are those based on community preferences, rather than patient or clinician ratings (Gold et al., 1996). Several methods are used to estimate quality-of-life weights. These include rating scale, standard gamble, time trade-off, and person trade-off approaches (Gold, Stevenson, and Fryback, 2002). Only the standard gamble approach is completely consistent with utility theory. However, the time trade-off method has also been widely applied in eliciting community preferences (Gold, Stevenson, and Fryback, 2002).

Quality-of-life weights can be directly elicited for individual specific health states or for a more general set of activity restrictions and health states that can then be used to construct QALY weights for specific conditions (Horsman et al., 2003; Kind, 1996). For this analysis, we used weights based on community-based preferences, using time trade-off or standard gamble when available. In some cases, we used patient or clinician ratings when no community preference-based weights were available. Sources for weights are discussed in more detail below. Table G-4 summarizes the key inputs for calculating QALYs associated with chronic health endpoints.

# G.5.1 Calculating QALYs Associated with Reductions in the Incidence of Chronic Bronchitis

CB is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 5 percent of the U.S. population (American Lung Association, 1999). For gains in quality of life resulting from reduced incidences of PM-induced CB, discounted QALYs are calculated as

DISCOUNTED QALY GAINED = 
$$\sum_{i} \Delta CB_{i} \times D_{i}^{*} \times (w_{i} - w_{i}^{CB})$$

# Table G-4. Summary of Key Parameters Used in QALY Calculations for ChronicDisease Endpoints

Parameter	Value(s)	Source(s)
Discount rate	0.03 (0.07 sensitivity analysis)	Gold et al. (1996), U.S. EPA (2000), U.S. OMB (2003)
Quality of life preference score for chronic bronchitis	0.5 – 0.7	Triangular distribution centered at 0.7 with upper bound at 0.9 (Vos, 1999a) (slightly better than a mild/moderate case) and a lower bound at 0.5 (average weight for a severe case based on Vos [1999a] and Smith and Peske [1994])
Duration of acute phase of acute myocardial infarction (AMI)	5.5 days – 22 days	Uniform distribution with lower bound based on average length of stay for an AMI (AHRQ, 2000) and upper bound based on Vos (1999b).
Probability of CHF post AMI	0.2	Vos, 1999a (WHO Burden of Disease Study, based on Cowie et al., 1997)
Probability of angina post AMI	0.51	American Heart Association, 2003 (Calculated as the population with angina divided by the total population with heart disease)
Quality-of-life preference score for post-AMI with CHF (no angina)	0.80 - 0.89	Uniform distribution with lower bound at 0.80 (Stinnett et al., 1996) and upper bound at 0.89 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality-of-life preference score for post-AMI with CHF and angina	0.76 – 0.85	Uniform distribution with lower bound at 0.76 (Stinnett et al., 1996, adjusted for severity) and upper bound at 0.85 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality-of-life preference score for post-AMI with angina (no CHF)	0.7 – 0.89	Uniform distribution with lower bound at 0.7, based on the standard gamble elicitation method (Pliskin, Stason, and Weinstein, 1981) and upper bound at 0.89, based on the time trade-off method (Kuntz et al., 1996).
Quality-of-life preference score for post-AMI (no angina, no CHF)	0.93	Only one value available from the literature. Thus, no distribution is specified. Source of value is Kuntz et al. (1996).

where  $\Delta CB_i$  is the number of incidences of CB avoided in age interval i,  $w_i$  is the average QALY weight for age interval i,  $w_i^{CB}$  is the QALY weight associated with CB,  $D_i^*$  is the discounted duration of life with CB for individuals with onset of disease in age interval i, equal to  $\int_{t=1}^{D_i} e^{-rt} dt$ , where  $D_i$  is the duration of life with CB for individuals with onset of disease of disease in age interval i.

A limited number of studies have estimated the impact of air pollution on new incidences of CB. Schwartz (1993) and Abbey et al. (1995) provide evidence that long-term PM exposure gives rise to the development of CB in the United States. Because this analysis focuses on the impacts of reducing ambient  $PM_{2.5}$ , only the Abbey et al. (1995) study is used, because it is the only study focusing on the relationship between  $PM_{2.5}$  and new incidences of CB. The number of cases of CB in each age interval is derived from applying the impact function from Abbey et al. (1995), to the population in each age interval with the appropriate baseline incidence rate.⁴ The effect estimate from the Abbey et al. (1995) study is 0.0137, which, based on the logistic specification of the model, is equivalent to a relative risk of 1.15 for a 10 µg change in  $PM_{2.5}$ . Table G-5 presents the estimated reduction in new incidences of CB associated with CAIR in 2010 and 2015.

CB is assumed to persist for the remainder of an affected individual's lifespan. Duration of CB will thus equal life expectancy conditioned on having CB. CDC has estimated that COPD (of which CB is one element) results in an average loss of life years equal to 4.26 per COPD death, relative to a reference life expectancy of 75 years (CDC, 2003). Thus, we subtract 4.26 from the remaining life expectancy for each age group, up to age 75. For age groups over 75, we apply the ratio of 4.26 to the life expectancy for the 65 to 74 year group (0.237) to the life expectancy for the 75 to 84 and 85 and up age groups to estimate potential life years lost and then subtract that value from the base life expectancy.

Quality of life with chronic lung diseases has been examined in several studies. In an analysis of the impacts of environmental exposures to contaminants, de Hollander et al. (1999) assigned a weight of 0.69 to years lived with CB. This weight was based on

⁴Prevalence rates for CB were obtained from the 1999 National Health Interview Survey (American Lung Association, 2002). Prevalence rates were available for three age groups: 18–44, 45–64, and 65 and older. Prevalence rates per person for these groups were 0.0367 for 18–44, 0.0505 for 45–64, and 0.0587 for 65 and older. The incidence rate for new cases of CB (0.00378 per person) was taken directly from Abbey et al. (1995).

	<b>Reduction in Incidence (95% Confidence Interval)</b>			
Age Interval	2010	2015		
25 - 34	1,100 (31 – 2,200)	1,400 (40 – 2,800)		
35 - 44	1,400 (41 – 2,800)	1,700 (47 – 3,300)		
45 - 54	1,600 (46 – 3,200)	1,900 (52 – 3,700)		
55 - 64	1,300 (36 – 2,500)	1,800 (49 – 3,400)		
65 - 74	770 (21 – 1,500)	1,100 (32 – 2,200)		
75 - 84	470 (13 – 910)	570 (16 - 1,100)		
85+	210 (6-400)	270 (8 - 530)		
Total	6,900 (190 – 14,000)	8,700 (240 - 17,000)		

 
 Table G-5. Estimated Reduction in Incidence of Chronic Bronchitis Associated with the Clean Air Interstate Rule

physicians' evaluations of health states similar to CB. Salomon and Murray (2003) estimated a pooled weight of 0.77 based on visual analogue scale, time trade-off, standard gamble, and person trade-off techniques applied to a convenience sample of health professionals. The Harvard Center for Risk Analysis catalog of preference scores reports a weight of 0.40 for severe COPD, with a range from 0.2 to 0.8, based on the judgments of the study's authors (Bell et al., 2001). The Victoria Burden of Disease (BoD) study used a weight of 0.47 for severe COPD and 0.83 for mild to moderate COPD, based on an analysis by Stouthard et al. (1997) of chronic diseases in Dutch populations (Vos, 1999a). Based on the recommendations of Gold et al. (1996), quality-of-life weights based on community preferences are preferred for CEA of interventions affecting broad populations. Use of weights based on health professionals is not recommended. It is not clear from the Victoria BoD study whether the weights used for COPD are based on community preferences or judgments of health professionals. The Harvard catalog score is clearly identified as based on author judgment. Given the lack of a clear preferred weight, we select a triangular distribution centered at 0.7 with an upper bound at 0.9 (slightly better than a mild/moderate case defined by the Victoria BoD study) and a lower bound at 0.5 based on the Victoria BoD study. We will need additional empirical data on quality of life with chronic respiratory diseases based on community preferences to improve our estimates.

Selection of a reference weight for the general population without CB is somewhat uncertain. It is clear that the general population is not in perfect health; however, there is some uncertainty as to whether individuals' ratings of health states are in reference to a perfect health state or to a generally achievable "normal" health state given age and general health status. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that "since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY" (Gold et al., 1996). Following Carrothers, Evans, and Graham (2002), we assumed that the reference weight for the general population without CB is 0.95. To allow for uncertainty in this parameter, we assigned a triangular distribution around this weight, bounded by 0.9 and 1.0. Note that the reference weight for the general population is used solely to determine the incremental quality-of-life improvement applied to the duration of life that would have been lived with the chronic disease. For example, if CB has a quality-of-life weight of 0.7 relative to a reference quality-of-life weight of 0.9, then the incremental quality-of-life improvement in 0.2. If the reference quality-of-life weight is 0.95, then the incremental quality-of-life improvement is 0.25. As noted above, the population is assumed to have a reference weight of 1.0 for all life years gained due to mortality risk reductions.

We present discounted QALYs over the duration of the lifespan with CB using a 3 percent discount rate. Based on the assumptions defined above, we used Monte Carlo simulation methods as implemented in the Crystal BallTM software program to develop the distribution of QALYs gained per incidence of CB for each age interval.⁵ Based on the assumptions defined above, the mean 3 percent discounted QALY gained per incidence of CB for each age interval resulting from the Monte Carlo simulation is presented in Table G-6. Table G-6 presents both the undiscounted and discounted QALYs gained per incidence.

# G.5.2 Calculating QALYs Associated with Reductions in the Incidence of Nonfatal Myocardial Infarctions

Nonfatal heart attacks, or acute myocardial infarctions, require more complicated calculations to derive estimates of QALY impacts. The actual heart attack, which results when an area of the heart muscle dies or is permanently damaged because of oxygen deprivation, and subsequent emergency care are of relatively short duration. Many heart

⁵Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables. For more details, see Gentile (1998).

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Age In	iterval	QALYs Gained per Incidence		
Start Age	End Age	Undiscounted	Discounted (3%)	
25	34	12.15 (4.40-19.95)	6.52 (2.36-10.71)	
35	44	9.91 (3.54-16.10)	5.94 (2.12-9.66)	
45	54	7.49 (2.71-12.34)	5.03 (1.82-8.29)	
55	64	5.36 (1.95-8.80)	4.03 (1.47-6.61)	
65	74	3.40 (1.22-5.64)	2.84 (1.02-4.71)	
75	84	2.15 (0.77-3.49)	1.92 (0.69-3.13)	
85+		0.79 (0.27-1.29)	0.77 (0.26-1.25)	

attacks result in sudden death. However, for survivors, the long-term impacts of advanced CHD are potentially of long duration and can result in significant losses in quality of life and life expectancy.

In this phase of the analysis, we did not independently estimate the gains in life expectancy associated with reductions in nonfatal heart attacks. Based on recommendations from the SAB-HES, we assumed that all gains in life expectancy are captured in the estimates of reduced mortality risk provided by the Pope et al. (2002) analysis. We only estimate the change in quality of life over the period of life affected by the occurrence of a heart attack. This may understate the QALY impacts of nonfatal heart attacks but ensures that the overall QALY impact estimates across endpoints do not double-count potential lifeyear gains.

Our approach adapts a CHD model developed for the Victoria Burden of Disease study (Vos, 1999b). This model accounts for the lost quality of life during the heart attack and the possible health states following the heart attack. Figure G-1 shows the heart attack QALY model in diagrammatic form. The total gain in QALYs is calculated as:

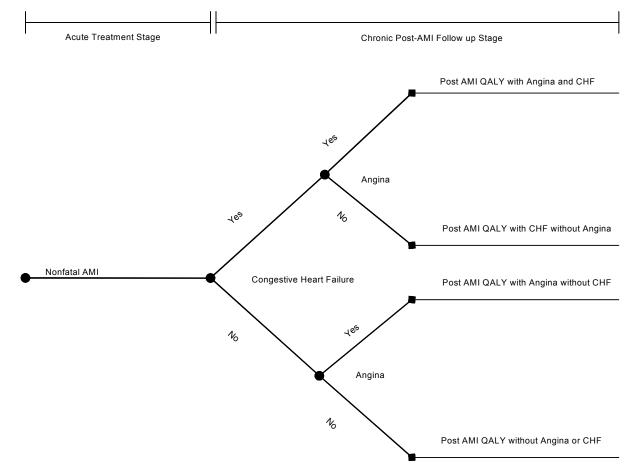


Figure G-1. Decision Tree Used in Modeling Gains in QALYs from Reduced Incidence of Nonfatal Acute Myocardial Infarctions

$$DISCOUNTED AMI QALY GAINED = \sum_{i} \Delta AMI_{i} \times D_{i}^{*AMI} \times (w_{i} - w_{i}^{AMI}) + \sum_{i} \sum_{j=1}^{4} \Delta AMI_{i} \times p_{j} D_{ij}^{*PostAMI} \times (w_{i} - w_{ij}^{PostAMI})$$

where  $\Delta AMI_i$  is the number of nonfatal acute myocardial infarctions avoided in age interval *i*,  $w_i^{AMI}$  is the QALY weight associated with the acute phase of the AMI,  $p_j$  is the probability of being in the *j*th post-AMI status,  $w_{ij}^{postAMI}$  is the QALY weight associated with post-AMI

health status *j*, w_i is the average QALY weight for age interval i,  $D_i^{*AMI} = \int_{t=1}^{D_i^{AMI}} e^{-rt} dt$ , the discounted value of  $D_i^{AMI}$ , the duration of the acute phase of the AMI, and  $D_i^{*postAMI} = \int_{t=1}^{D_i^{postAMI}} e^{-rt} dt$ , is the discounted value of  $D_{ij}^{PostAMI}$ , the duration of post-AMI

health status *j*.

Nonfatal heart attacks have been linked with short-term exposures to PM25 in the United States (Peters et al., 2001) and other countries (Poloniecki et al., 1997). We used a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between PM_{2.5} and nonfatal heart attacks. Peters et al. is the only available U.S. study to provide a specific estimate for heart attacks. Other studies, such as Samet et al. (2000) and Moolgavkar (2000), show a consistent relationship between all cardiovascular hospital admissions, including for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on longer-term health costs and earnings, we chose to provide a separate estimate for nonfatal heart attacks based on the single available U.S. effect estimate. The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the United States. These studies provide a weight of evidence for this type of effect. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other CHDs (Carthenon et al., 2002; Dekker et al., 2000; Liao et al., 1997, Tsuji et al., 1996). As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

The number of avoided nonfatal AMI in each age interval is derived from applying the impact function from Peters et al. (2001) to the population in each age interval with the appropriate baseline incidence rate.⁶ The effect estimate from the Peters et al. (2001) study is 0.0241, which, based on the logistic specification of the model, is equivalent to a relative

⁶Daily nonfatal myocardial infarction incidence rates per person were obtained from the 1999 National Hospital Discharge Survey (assuming all diagnosed nonfatal AMI visit the hospital). Age-specific rates for four regions are used in the analysis. Regional averages for populations 18 and older are 0.0000159 for the Northeast, 0.0000135 for the Midwest, 0.0000111 for the South, and 0.0000100 for the West.

risk of 1.27 for a 10  $\mu$ g change in PM_{2.5}. Table G-7 presents the estimated reduction in nonfatal AMI associated with CAIR in 2010 and 2015.

	Reduction in Incidence*(95% Confidence Interval)		
Age Interval	2010	2015	
18-24	9 (2 - 16)	11 (3 – 18)	
25 - 34	92 (23 - 160)	120 (29 – 200)	
35 - 44	630 (160 - 1,100)	700 (180 – 1,200)	
45 - 54	2,400 (600 - 4,200)	2,800 (690 - 4,800)	
55 - 64	4,000 (990 - 6,900)	5,300 (1,300 – 9,200)	
65 - 74	4,000 (990 - 6,900)	5,800 (1,400 - 10,000)	
75 - 84	3,800 (940 - 6,500)	4,600 (1,100 - 7,900)	
85+	2,300 (570 - 4,000)	3,000 (750 - 5,200)	
Total	17,000 (4,300 - 30,000)	22,000 (5,600 - 39,000)	

 Table G-7. Estimated Reduction in Nonfatal Acute Myocardial Infarctions Associated

 with the Clean Air Interstate Rule

Acute myocardial infarction results in significant loss of quality of life for a relatively short duration. The WHO Global Burden of Disease study, as reported in Vos (1999b), assumes that the acute phase of an acute myocardial infarction lasts for 0.06 years, or around 22 days. An alternative assumption is the acute phase is characterized by the average length of hospital stay for an AMI in the United States, which is 5.5 days, based on data from the Agency for Healthcare Research and Quality's Healthcare Cost and Utilization Project (HCUP).⁷ We assumed a distribution of acute phase duration characterized by a uniform distribution between 5.5 and 22 days, noting that due to earlier discharges and in-home therapy available in the United States, duration of reduced quality of life may continue after discharge from the hospital. In the period during and directly following an AMI (the acute phase), we assigned a quality of life weight equal to 0.605, consistent with the weight for the period in treatment during and immediately after an attack (Vos, 1999b).

⁷Average length of stay estimated from the HCUP data includes all discharges, including those due to death. As such, the 5.5-day average length of stay is likely an underestimate of the average length of stay for AMI admissions where the patient is discharged alive.

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During the post-AMI period, a number of different health states can determine the loss in quality of life. We chose to classify post-AMI health status into four states defined by the presence or absence of angina and congestive heart failure (CHF). This makes a very explicit assumption that without the occurrence of an AMI, individuals would not experience either angina or CHF. If in fact individuals already have CHF or angina, then the quality of life gained will be overstated. We do not have information about the percentage of the population have been diagnosed with angina or CHF with no occurrence of an AMI. Nor do we have information on what proportion of the heart attacks occurring due to PM exposure are first heart attacks versus repeat attacks. Probabilities for the four post-AMI health states sum to one.

Given the occurrence of a nonfatal AMI, the probability of congestive heart failure is set at 0.2, following the heart disease model developed by Vos (1999b). The probability is based on a study by Cowie et al. (1997), which estimated that 20 percent of those surviving AMI develop heart failure, based on an analysis of the results of the Framingham Heart Study.

The probability of angina is based on the prevalence rate of angina in the U.S. population. Using data from the American Heart Association, we calculated the prevalence rate for angina by dividing the estimated number of people with angina (6.6 million) by the estimated number of people with CHD of all types (12.9 million). We then assumed that the prevalence of angina in the population surviving an AMI is similar to the prevalence of angina in the total population with CHD. The estimated prevalence rate is 51 percent, so the probability of angina is 0.51.

Combining these factors leads to the probabilities for each of the four health states as follows:

- I. Post AMI with CHF and angina = 0.102
- II. Post AMI with CHF without angina = 0.098
- III. Post AMI with angina without CHF = 0.408
- IV. Post AMI without angina or CHF = 0.392

Duration of post-AMI health states varies, based in part on assumptions regarding life expectancy with post-AMI complicating health conditions. Based on the model used for established market economies (EME) in the WHO Global Burden of Disease study, as reported in Vos (1999b), we assumed that individuals with CHF have a relatively short

remaining life expectancy and thus a relatively short period with reduced quality of life (recall that gains in life expectancy are assumed to be captured by the cohort estimates of reduced mortality risk). Table G-8 provides the duration (both discounted and undiscounted) of CHF assumed for post-AMI cases by age interval.

Age Interval		<b>Duration of Heart Failure</b>	
Start Age	End Age	Undiscounted	Discounted (3%)
18	24	7.11	6.51
25	34	6.98	6.40
35	44	6.49	6.00
45	54	5.31	4.99
55	64	1.96	1.93
65	74	1.71	1.69
75	84	1.52	1.50
85+		1.52	1.50

Table G-8. Assumed Duration of Congestive Heart Failure

Duration of health states without CHF is assumed to be equal to the life expectancy of individuals conditional on surviving an AMI. Ganz et al. (2000) note that "Because patients with a history of myocardial infarction have a higher chance of dying of CHD that is unrelated to recurrent myocardial infarction (for example, arrhythmia), this cohort has a higher risk for death from causes other than myocardial infarction or stroke than does an unselected population." They go on to specify a mortality risk ratio of 1.52 for mortality from other causes for the cohort of individuals with a previous (nonfatal) AMI. The risk ratio is relative to all-cause mortality for an age-matched unselected population (i.e., general population). We adopted the same ratios and applied them to each age-specific all-cause mortality rate to derive life expectancies (both discounted and undiscounted) for each age group after an AMI, presented in Table G-9. These life expectancies are then used to represent the duration of non-CHF post-AMI health states (III and IV).

For the four post-AMI health states, we used QALY weights based on preferences for the combined conditions characterizing each health state. A number of estimates of QALY weights are available for post-AMI health conditions.

Age Ir	Age Interval		pectancy (non-CHF)
Start Age	End Age	Undiscounted	Discounted (3%)
18	24	55.5	27.68
25	34	46.1	25.54
35	44	36.8	22.76
45	54	27.9	19.28
55	64	19.8	15.21
65	74	12.8	10.82
75	84	7.4	6.75
85+		3.6	3.47

Table G-9. Assumed Duration of Non-CHF Post-AMI Health States

The first two health states are characterized by the presence of CHF, with or without angina. The Harvard Center for Risk Analysis catalog of preference scores provides several specific weights for CHF with and without mild or severe angina and one set specific to post-AMI CHF. Following the Victoria Burden of Disease model, we assumed that most cases of angina will be treated and thus kept at a mild to moderate state. We thus focused our selection on QALY weights for mild to moderate angina. The Harvard database includes two sets of community preference-based scores for CHF (Stinnett et al., 1996; Kuntz et al., 1996). The scores for CHF with angina range from 0.736 to 0.85. The lower of the two scores is based on angina in general with no delineation by severity. Based on the range of the scores for mild to severe cases of angina in the second study, one can infer that an average case of angina has a score around 0.96 of the score for a mild case. Applying this adjustment raises the lower end of the range of preference scores for a mild case of angina to 0.76. We selected a uniform distribution over the range 0.76 to 0.85 for CHF with mild angina, with a midpoint of 0.81. The same two studies in the Harvard catalog also provide weights for CHF without angina. These scores range from 0.801 to 0.89. We selected a uniform distribution over this range, with a midpoint of 0.85.

The third health state is characterized by angina, without the presence of CHF. The Harvard catalog includes five sets of community preference-based scores for angina, one that specifies scores for both mild and severe angina (Kuntz et al., 1996), one that specifies mild angina only (Pliskin, Stason, and Weinstein, 1981), one that specifies severe angina only (Cohen, Breall, and Ho, 1994), and two that specify angina with no severity classification

(Salkeld, Phongsavan, and Oldenburg, 1997; Stinnett et al., 1996). With the exception of the Pliskin, Stason, and Weinstein score, all of the angina scores are based on the time trade-off method of elicitation. The Pliskin, Stason, and Weinstein score is based on the standard gamble elicitation method. The scores for the nonspecific severity angina fall within the range of the two scores for mild angina specifically. Thus, we used the range of mild angina scores as the endpoints of a uniform distribution. The range of mild angina scores is from 0.7 to 0.89, with a midpoint of 0.80.

For the fourth health state, characterized by the absence of CHF and/or angina, there is only one relevant community preference score available from the Harvard catalog. This score is 0.93, derived from a time trade-off elicitation (Kuntz et al., 1996). Insufficient information is available to provide a distribution for this weight; therefore, it is treated as a fixed value.

Similar to CB, we assumed that the reference weight for the general population without AMI is 0.95. To allow for uncertainty in this parameter, we assigned a triangular distribution around this weight, bounded by 0.9 and 1.0.

Based on the assumptions defined above, we used Monte Carlo simulation methods as implemented in the Crystal BallTM software program to develop the distribution of QALYs gained per incidence of nonfatal AMI for each age interval. For the Monte Carlo simulation, all distributions were assumed to be independent. The mean QALYs gained per incidence of nonfatal AMI for each age interval is presented in Table G-10, along with the 95 percent confidence interval resulting from the Monte Carlo simulation. Table G-10 presents both the undiscounted and discounted QALYs gained per incidence.

# G.6 Cost-Effectiveness Analysis

Given the estimates of changes in life expectancy and quality of life, the next step is to aggregate life expectancy and quality-of-life gains to form an effectiveness measure that can be compared to costs to develop cost-effectiveness ratios. This section discusses the proper characterization of the combined effectiveness measure and the appropriate calculation of the numerator of the cost-effectiveness ratio.

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 Table G-10. QALYs Gained per Avoided Nonfatal Myocardial Infarction

Age Interval		QALYs Gained per Incidence ^a	
Start Age	End Age	Undiscounted	Discounted (3%)
18	24	4.18 (1.24-7.09)	2.17 (0.70-3.62)
25	34	3.48 (1.09-5.87)	2.00 (0.68-3.33)
35	44	2.81 (0.88-4.74)	1.79 (0.60-2.99)
45	54	2.14 (0.67-3.61)	1.52 (0.51-2.53)
55	64	1.49 (0.42-2.52)	1.16 (0.34-1.95)
65	74	0.97 (0.30-1.64)	0.83 (0.26-1.39)
75	84	0.59 (0.20-0.97)	0.54 (0.19-0.89)
85+		0.32 (0.13-0.50)	0.31 (0.13-0.49)

^a Mean of Monte Carlo generated distribution; 95% confidence interval presented in parentheses.

# G.6.1 Aggregating Life Expectancy and Quality-of-Life Gains

To develop an integrated measure of changes in health, we simply sum together the gains in life years from reduced mortality risk in each age interval with the gains in QALYs from reductions in incidence of CB and acute myocardial infarctions. The resulting measure of effectiveness then forms the denominator in the cost-effectiveness ratio. What is this combined measure of effectiveness? It is not a QALY measure in a strict sense, because we have not adjusted life-expectancy gains for preexisting health status (quality of life). It is however, an effectiveness measure that adds to the standard life years calculation a scaled morbidity equivalent. Thus, we term the aggregate measure morbidity inclusive life years, or MILYs. Alternatively, the combined measure could be considered as QALYs with an assumption that the community preference weight for all life-expectancy gains is 1.0. If one considers that this weight might be considered to be a "fair" treatment of those with

preexisting disabilities, the effectiveness measure might be termed "fair QALY" gained. However, this implies that all aspects of fairness have been addressed, and there are clearly other issues with the fairness of QALYs (or other effectiveness measures) that are not addressed in this simple adjustment. The MILY measure violates some of the properties used in deriving QALY weights, such as linear substitution between quality of life and quantity of life. However, in aggregating life expectancy and quality-of-life gains, it merely represents an alternative social weighting that is consistent with the spirit of the recent OMB guidance on CEA. The guidance notes that "fairness is important in the choice and execution of effectiveness measures" (OMB, 2003). The resulting aggregate measure of effectiveness will not be consistent with a strict utility interpretation of QALYs; however, it may still be a useful index of effectiveness.

Applying the life expectancies and distributions of QALYs per incidence for CB and AMI to estimated distributions of incidences yields distributions of life expectancy and QALYs gained due to CAIR. These distributions reflect both the quantified uncertainty in incidence estimates and the quantified uncertainty in QALYs gained per incidence.

For the CAIR 2010 analysis year, Table G-11 presents the mean 3 percent discounted MILYs gained for each age interval, broken out by life expectancy and quality-of-life categories. Note that quality-of-life gains occur from age 18 and up, while life expectancy gains accrue only after age 29. This is based on the ages of the study populations in the underlying epidemiological studies. It is unlikely that such discontinuities exist in reality, but to avoid overstating effectiveness, we chose to limit the life-expectancy gains to those occurring in the population 30 and over and the morbidity gains to the specific adult populations examined in the studies. Table G-12 provides the same information for the 2015 analysis year.

It is worth noting that around a third of mortality-related benefits are due to reductions in premature deaths among those 75 and older, while only 7 percent of morbidity benefits occur in this age group. This is due to two factors: (1) the relatively low baseline mortality rates in populations under 75, and (2) the relatively constant baseline rates of chronic disease coupled with the relatively long period of life that is lived with increased quality of life without CB and advanced heart disease.

Age	Life Years Gained from Mortality Risk Reductions (95% CI)	QALY Gained from Reductions in Chronic Bronchitis (95% CI)	QALY Gained from Reductions in Acute Myocardial Infarctions (95% CI)	Total Gain in MILYs (95% CI)
18–24	_	—	20 (4 - 42)	20 (4 - 42)
25–34	2,500	7,300	180	10,000
	(880 – 4,100)	(430 – 18,000)	(34 – 410)	(3,000 – 20,000)
35–44	9,100	8,500	1,100	19,000
	(3,100 – 15,000)	(330 – 21,000)	(200 – 2,500)	(8,200 – 32,000)
45–54	19,000	8,200	3,600	31,000
	(6,600 – 31,000)	(410 – 20,000)	(640 - 8,100)	(15,000 – 48,000)
55–64	31,000	5,200	4,500	41,000
	(11,000 – 51,000)	(310 – 13,000)	(760 – 10,000)	(19,000 – 63,000)
65–74	34,000	2,200	3,200	40,000
	(12,000 – 57,000)	(110 – 5,100)	(590 – 7,300)	(17,000 – 63,000)
75–84	33,000	900	2,000	36,000
	(11,000 – 54,000)	(43 – 2,100)	(380 – 4,400)	(14,000 – 57,000)
85+	15,000	160	680	16,000
	(5,200 – 25,000)	(7 – 370)	(130 – 1,500)	(6,000 – 26,000)
Total	140,000	33,000	15,000	190,000
	(100,000 – 180,000)	(1,700 – 78,000)	(2,900 – 34,000)	(140,000 – 250,000)

 Table G-11. Estimated Gains in 3 Percent Discounted MILYs Associated with the

 Clean Air Interstate Rule in 2010^a

^a Note that all estimates have been rounded to two significant digits.

The relationship between age and the distribution of MILYs gained from mortality and morbidity is shown in Figure G-2. Because the baseline mortality rate is increasing in age at a much faster rate than the prevalence rate for CB, the share of MILYs gained accounted for by mortality is proportional to age. At the oldest age interval, avoiding incidences of CB leads to only a few MILYs gained, due to the lower number of years lived with CB. MILYs gained from avoided premature mortality is low in the youngest age intervals because of the low overall mortality rates in these intervals, although the number of MILYs per incidence is high. In later years, even though the MILYs gained per incidence avoided is low, the number of cases is very high due to higher baseline mortality rates.

Age	Life Years Gained from Mortality Risk Reductions (95% CI)	QALY Gained from Reductions in Chronic Bronchitis (95% CI)	QALY Gained from Reductions in Acute Myocardial Infarctions (95% CI)	Total Gain in MILYs (95% CI)
18–24			22 (4 - 52)	22 (4 - 52)
25–34	3,200	9,400	230	13,000
	(1,100 – 5,300)	(510 – 22,000)	(42 – 520)	(3,800 – 26,000)
35–44	11,000	9,800	1,250	22,000
	(3,800 – 17,000)	(670 – 24,000)	(230 – 2,800)	(9,600 – 37,000)
45–54	22,000	9,400	4,100	36,000
	(8,200 – 36,000)	(430 – 23,000)	(780 – 9,300)	(18,000 – 55,000)
55–64	42,000	7,100	5,900	55,000
	(15,000 – 69,000)	(440 – 17,000)	(1,000 – 14,000)	(27,000 – 84,000)
65–74	51,000	3,200	4,700	59,000
	(18,000 – 84,000)	(230 – 7,800)	(870 – 11,000)	(26,000 – 92,000)
75–84	40,000	1,100	2,400	44,000
	(15,000 – 65,000)	(68 – 2,700)	(430 -5,400)	(18,000 – 69,000)
85+	20,000	210	890	21,000
	(7,400 – 33,000)	(14 – 500)	(180 – 1,900)	(8,400 - 34,000)
Total	190,000	40,000	20,000	250,000
	(140,000 – 240,000)	(2,400 – 96,000)	(3,600 - 44,000)	(180,000 - 330,000)

 Table G-12. Estimated Gains in 3 Percent Discounted MILYs Associated with the

 Clean Air Interstate Rule in 2015^a

^a Note that all estimates have been rounded to two significant digits.

Summing over the age intervals provides estimates of total MILYs gained for CAIR in 2010 and 2015. The total number of discounted (3 percent) MILYs gained in 2010 is 190,000 (95% CI: 140,000 – 250,000) and in 2015 is 250,000 (95% CI: 180,000 – 330,000).

# G.6.2 Dealing with Acute Health Effects and Nonhealth Effects

Health effects from exposure to particulate air pollution encompass a wide array of chronic and acute conditions in addition to premature mortality (EPA, 1996). Although chronic conditions and premature mortality generally account for the majority of monetized benefits, acute symptoms can affect a broad population or sensitive populations (e.g., asthma exacerbations in asthmatic children. In addition, reductions in air pollution may result in a

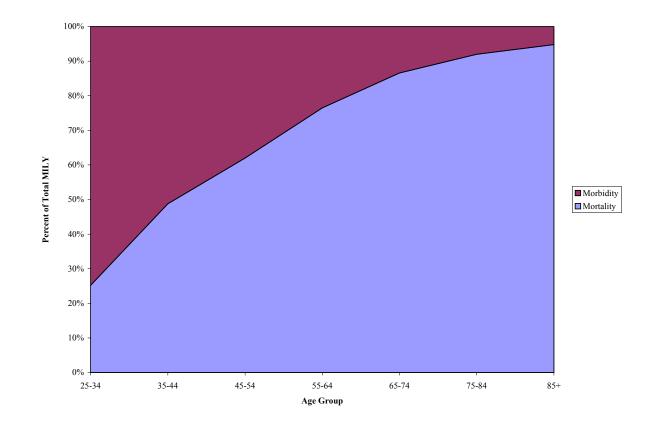


Figure G-2. Distribution of Mortality and Morbidity Related MILY Across Age Groups for the CAIR in 2015 (3 percent Discount Rate)

broad set of nonhealth environmental benefits, including improved visibility in national parks, increased agricultural and forestry yields, reduced acid damage to buildings, and a host of other impacts. QALYs address only health impacts, and the OMB guidance notes that "where regulation may yield several different beneficial outcomes, a cost-effectiveness comparison becomes more difficult to interpret because there is more than one measure of effectiveness to incorporate in the analysis."

With regard to acute health impacts, Bala and Zarkin (2000) suggest that QALYs are not appropriate for valuing acute symptoms, because of problems with both measuring utility for acute health states and applying QALYs in a linear fashion to very short duration health states. Johnson and Lievense (2000) suggest using conjoint analysis to get healthy-utility time equivalences that can be compared across acute effects, but it is not clear how these can be combined with QALYs for chronic effects and loss of life expectancy. There is also a class of effects that EPA has traditionally treated as acute, such as hospital admissions, which may also result in a loss of quality of life for a period of time following the effect. For example, life after asthma hospitalization has been estimated with a utility weight of 0.93 (Bell et al., 2001; Kerridge, Glasziou, and Hillman, 1995).

How should these effects be combined with QALYs for chronic and mortality effects? One method would be to convert the acute effects to QALYs; however, as noted above, there are problems with the linearity assumption (i.e., if a year with asthma symptoms is equivalent to 0.7 year without asthma symptoms, then 1 day without asthma symptoms is equivalent to 0.0019 QALY gained). This is troubling from both a conceptual basis and a presentation basis. An alternative approach is simply to treat acute health effects like nonhealth benefits and subtract the dollar value (based on WTP or COI) from compliance costs in the CEA.

To address the issues of incorporating acute morbidity and nonhealth benefits, OMB suggests that agencies "subtract the monetary estimate of the ancillary benefits from the gross cost estimate to yield an estimated net cost." As with benefit-cost analysis, any unquantified benefits and/or costs should be noted and an indication of how they might affect the cost-effectiveness ratio should be described. We will follow this recommended "net cost" approach in the illustrative exercise, specifically in netting out the benefits of health improvements other than reduced mortality and chronic morbidity, and the benefits of improvements in visibility at national parks (see Chapter 4 for more details on these benefit categories).

# G.6.3 Cost-Effectiveness Ratios

Construction of cost-effectiveness ratios requires estimates of effectiveness (in this case measured by lives saved, life years gained, or MILYs gained) in the denominator and estimates of costs in the numerator. The estimate of costs in the numerator should include both the direct costs of the controls necessary to achieve the reduction in ambient PM_{2.5} and the avoided costs (cost savings) associated with the reductions in morbidity (Gold et al., 1996). In general, because reductions in air pollution do not require direct actions by the affected populations, there are no specific costs to affected individuals (aside from the overall increases in prices that might be expected to occur as control costs are passed on by affected industries). Likewise, because individuals do not engage in any specific actions to realize the health benefit of the pollution reduction, there are no decreases in utility (as might occur from a medical intervention) that need to be adjusted for in the denominator. Thus, the

elements of the numerator are direct costs of controls minus the avoided COI associated with CB and nonfatal AMI. In addition, to account for the value of reductions in acute health impacts and nonhealth benefits, we net out the monetized value of these benefits from the numerator to yield a "net cost" estimate. For the MILY aggregate effectiveness measure, the denominator is simply the sum of life years gained from increased life expectancy and the sum of QALYs gained from the reductions in CB and nonfatal AMI.

Avoided costs for CB and nonfatal AMI are based on estimates of lost earnings and medical costs.⁸ Using age-specific annual lost earnings and medical costs estimated by Cropper and Krupnick (1990) and a 3 percent discount rate, we estimated a lifetime present discounted value (in 2000\$) due to CB of \$150,542 for someone between the ages of 27 and 44; \$97,610 for someone between the ages of 45 and 64; and \$11,088 for someone over 65. The corresponding age-specific estimates of lifetime present discounted value (in 2000\$) using a 7 percent discount rate are \$86,026, \$72,261, and \$9,030, respectively. These estimates assumed that 1) lost earnings continue only until age 65, 2) medical expenditures are incurred until death, and 3) life expectancy is unchanged by CB.

Because the costs associated with a myocardial infarction extend beyond the initial event itself, we consider costs incurred over several years. Using age-specific annual lost earnings estimated by Cropper and Krupnick (1990) and a 3 percent discount rate, we estimated a present discounted value in lost earnings (in 2000\$) over 5 years due to a myocardial infarction of \$8,774 for someone between the ages of 25 and 44, \$12,932 for someone between the ages of 45 and 54, and \$74,746 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2000\$) using a 7 percent discount rate are \$7,855, \$11,578, and \$66,920, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. Thus, we do not include lost earnings in the cost estimates for these age groups.

Two estimates of the direct medical costs of myocardial infarction are used. The first estimate is from Wittels, Hay, and Gotto (1990), which estimated expected total medical costs of MI over 5 years to be \$51,211 (in 1986\$) for people who were admitted to the

⁸Gold et al. (1996) recommend not including lost earnings in the cost-of-illness estimates, suggesting that in some cases, they may be already be counted in the effectiveness measures. However, this requires that individuals fully incorporate the value of lost earnings and reduced labor force participation opportunities into their responses to time-tradeoff or standard-gamble questions. For the purposes of this analysis and for consistency with the way costs-of-illness are calculated for the benefit-cost analysis, we have assumed that individuals do not incorporate lost earnings in responses to these questions. This assumption can be relaxed in future analyses with improved understanding of how lost earnings are treated in preference elicitations.

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hospital and survived hospitalization (there does not appear to be any discounting used). Using the CPI-U for medical care, the Wittels estimate is \$109,474 in year 2000\$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes, and prices (using "knowledgeable cardiologists" as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The second estimate is from Russell et al. (1998), which estimated first-year direct medical costs of treating nonfatal myocardial infarction of \$15,540 (in 1995\$), and \$1,051 annually thereafter. Converting to year 2000\$, that would be \$23,353 for a 5-year period (without discounting).

The two estimates from these studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used estimates for medical costs that similarly cover a 5-year period. We used a simple average of the two 5-year estimates, or \$65,902, and add it to the 5-year opportunity cost estimate. The resulting estimates are given in Table G-13.

Age Group	<b>Opportunity Cost</b>	Medical Cost ^a	Total Cost
0 - 24	\$0	\$65,902	\$65,902
25-44	\$8,774 ^b	\$65,902	\$74,676
45 - 54	\$12,253 ^b	\$65,902	\$78,834
55 - 65	\$70,619 ^b	\$65,902	\$140,649
>65	\$0	\$65,902	\$65,902

 Table G-13. Estimated Costs Over a 5-Year Period (in 2000\$) of a Nonfatal Myocardial Infarction

^a An average of the 5-year costs estimated by Wittels, Hay, and Gotto (1990) and Russell et al. (1998).

^b From Cropper and Krupnick (1990), using a 3 percent discount rate.

The total avoided COI by age group associated with the reductions in CB and nonfatal acute myocardial infarctions is provided in Table G-14. Note that the total avoided COI associated with CAIR is \$2.1 billion in 2010 and \$2.7 billion in 2015. Note that this does not include any direct avoided medical costs associated with premature mortality. Nor does it include any medical costs that occur more than 5 years from the onset of a nonfatal AMI. Therefore, this is likely an underestimate of the true avoided COI associated with CAIR.

	Avoided Cost of Illness (in millions of 2000\$) (95% confidence interval) ^a			
Γ	Chronic	Bronchitis	Nonfatal Acute Myocardial Infarction	
Age Range	2010	2015	2010	2015
18-24	—	—	\$1 (\$0-\$2)	\$1 (\$0-\$2)
25-34	\$166	\$212	\$7	\$8
	(\$5 - \$325)	(\$6 - \$415)	(\$1-\$17)	(\$1-\$22)
35-44	\$215	\$247	\$46	\$52
	(\$6-\$421)	(\$7-\$484)	(\$7 – \$118)	(\$8 - \$134)
45-54	\$156	\$179	\$184	\$211
	(\$4 - \$305)	(\$5 - \$350)	(\$29 – \$467)	(\$33 - \$535)
55-64	\$125	\$168	\$547	\$728
	(\$3 – \$244)	(\$5 - \$328)	(\$133 – \$1,171)	(\$177 – \$1,558)
65-74	\$8	\$12	\$251	\$367
	(\$0-\$16)	(\$0-\$24)	(\$30 – \$684)	(\$44 – \$999)
75-84	\$5	\$6	\$238	\$289
	(\$0-\$10)	(\$0-\$12)	(\$28 – \$649)	(\$34 – \$788)
85+	\$2	\$3	\$145	\$190
	(\$0-\$4)	(\$0-\$6)	(\$17 – \$396)	(\$23 - \$516)
Total	\$677	\$827	\$1,418	\$1,846
	(\$360 – \$991)	(\$447 – \$1,213)	(\$680 – \$2,310)	(\$870 – \$3,007)

 Table G-14. Avoided Costs of Illness Associated with Reductions in Chronic Bronchitis and Nonfatal Acute Myocardial Infarctions Associated with CAIR in 2010 and 2015

Note that the confidence intervals for avoided COI include both the uncertainty in the unit values for each health effect and the uncertainty in the estimated change in incidence for each health effect. Uncertainties are combined using Monte Carlo simulation methods.

In a traditional cost-effectiveness analysis, net costs of the intervention would be divided by the effectiveness measure to calculate a cost per life year or cost per QALY. However, for both of the years of analysis, net costs of CAIR are negative, implying that CAIR is a cost-saving rule. However, it is possible to calculate the costs that would be necessary for the cost-effectiveness of CAIR to exceed various thresholds. Costeffectiveness ratios are usually interpreted in a relative sense, because there is no universally agreed on cost-effectiveness cutoff for environmental health interventions. Although the U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine did not recommend a cost-effectiveness threshold for generalized use, it may be useful to identify cost thresholds that would make CAIR cost-ineffective relative to other life-saving or quality of life-improving interventions. The Harvard Cost Utility Analysis database suggests a median cost-utility ratio of \$31,000 per QALY (2002\$) for respiratory and cardiovascular interventions, while Teng et al. (1995) report a median cost per life-year saved for live-saving interventions of \$48,000 (1993\$). The health economics literature often uses either \$50,000 or \$100,000 per QALY as a de facto cut point with ratios less than these values considered cost-effective. For the purposes of this analysis, we computed the costs necessary to exceed the \$50,000 and \$100,000 cost-effectiveness thresholds, without endorsing \$50,000 or \$100,000 as an absolute threshold beyond which interventions should not be implemented. Decisions as to whether a specific control strategy is justified should be based on a complete comparison of benefits and costs.

Table G-15 summarizes the effectiveness measures and avoided costs associated with CAIR in 2010 and 2015 and presents the implicit costs of CAIR that would be necessary for the cost-effectiveness ratio to exceed the \$50,000 and \$100,000 threshold.

# G.7 Discount Rate Sensitivity Analysis

A large number of parameters and assumptions are necessary in conducting a CEA. Where appropriate and supported by data, we have included distributions of parameter values that were used in generating the reported confidence intervals. For the assumed discount rate, we felt it more appropriate to examine the impact of the assumption using a sensitivity analysis rather than through the integrated probabilistic uncertainty analysis.

The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the academic community. OMB and EPA guidance require using both a 7 percent rate and a 3 percent rate. In the most recent benefit-cost analyses of air pollution regulations, a 3 and 7 percent discount rate have been adopted in the primary analysis. A 3 percent discount rate reflects a "social rate of time preference" discounting concept. A 3 percent discount rate is also consistent with the recommendations of the NAS panel on CEA (Gold et al., 1996), which suggests that "a real annual (riskless) rate of 3 percent should be used in the Reference Case analysis." We have also calculated MILYs and the implicit cost thresholds using a 7 percent rate consistent with an "opportunity cost of capital" concept to reflect the time value of resources directed to meet regulatory requirements. Further discussion of this topic appears in Chapter 7 of Gold et al. (1996), in Chapter 6 of the EPA *Guidelines for Economic Analysis*, and in OMB Circular A-4.

	Result Using 3% Discount Rate (95% Confidence Interval)		
	2010	2015	
Life years gained from mortality risk reductions	140,000 (100,000 – 180,000)	190,000 (140,000 – 240,000)	
QALY gained from reductions in chronic bronchitis	33,000 (1,700 – 78,000)	40,000 (2,400 - 96,000)	
QALY gained from reductions in acute myocardial infarctions	15,000 (2,900 – 34,000)	20,000 (3,600 - 44,000)	
Total gain in MILYs	190,000 (140,000 – 250,000)	250,000 (180,000 - 330,000)	
Avoided cost of illness			
Chronic bronchitis	\$680 million (\$360 million – \$990 million)	\$830 million (\$450 million – \$1,200 million)	
Nonfatal AMI	\$1,400 million (\$680 million – \$2,300 million)	\$1,800 million (\$870 million – \$3,000 million)	
CAIR rule costs ^b	\$2.4 billion	\$3.6 billion	
Net cost per MILYs	Cost saving	Cost saving	
Implied annual cost necessary to exceed \$50,000/QALY threshold	\$13 billion (\$10 billion – \$18 billion)	\$18 billion (\$13 billion – \$23 billion)	
Implied annual cost necessary to exceed \$100,000/QALY threshold	\$23 billion (\$17 billion – \$31 billion)	\$30 billion (\$22 billion – \$40 billion)	

#### Table G-15. Summary of Results for the Clean Air Interstate Rule^a

^a Consistent with recommendations of Gold et al. (1996), all summary results are reported at a precision level of two significant digits to reflect limits in the precision of the underlying elements.

^b Costs are the private firm costs of compliance derived from the Integrated Planning Model, as discussed in Chapter 2, and reflect discounting using firm specific costs of capital.

Table G-16 presents a summary of results using the 7 percent discount rate and the percentage difference between the 7 percent results and the base case 3 percent results. Adoption of a 7 percent discount rate decreases the estimated life years and QALYs gained from implementing the CAIR. Adopting a discount rate of 7 percent results in a 26 percent reduction in the estimated total MILYs gained in each year, while the implicit cost necessary to exceed the \$50,000 cost-effectiveness threshold is reduced by 20 percent in each year.

**Percentage Change Result Using 7 Percent Relative to Result Using 3 Discount Rate Percent Discount Rate** -25Life years gained from mortality 140.000 risk reductions QALY gained from reductions in 26,000 -35chronic bronchitis QALY gained from reductions in 15,000 -22acute myocardial infarctions Total gain in MILYs 180,000 -26Avoided cost of illness Chronic bronchitis \$540 million -35\$1,800 million Nonfatal AMI  $-3^{a}$ Implied cost necessary to exceed \$14 billion -20\$50,000/QALY threshold \$24 billion -23 Implied cost necessary to exceed

Table G-16. Impacts of Using a 7 Percent Discount Rate on Cost Effectiveness Analysisfor the Clean Air Interstate Rule in 2015

^a There is a 3 percent difference in estimated avoided costs of nonfatal AMI; however, because of rounding, the reported cost for both 3 and 7 percent discount rates is \$1,800 million.

# G.8 Conclusions

\$100,000/QALY threshold

We calculated the effectiveness of CAIR based on reductions in premature deaths and incidence of chronic disease. We measured effectiveness using several different metrics, including lives saved, life years saved, and QALYs (for improvements in quality of life due to reductions in incidence of chronic disease). We suggested a new metric for aggregating life years saved and improvements in quality of life, morbidity inclusive life years (MILY) which assumes that society assigns a weight of one to years of life extended regardless of preexisting disabilities or chronic health conditions.

Using the MILYs metric, we estimated that CAIR could cost up to \$14 billion annually in 2010 and up to \$18 billion annually in 2015 and would still likely be cost-

effective relative to other health interventions for cardiovascular and respiratory disease. Given costs of \$2.4 billion and \$3.6 billion in 2010 and 2015, respectively, CAIR is clearly a very cost-effective way to achieve improvements in public health.

CEA of environmental regulations that have substantial public health impacts may be informative in identifying programs that have achieved cost-effective reductions in health impacts and can suggest areas where additional controls may be justified. However, the overall efficiency of a regulatory action can only be judged through a complete benefit-cost analysis that takes into account all benefits and costs, including both health and nonhealth effects. The benefit-cost analysis for CAIR, provided in Chapter 4, shows that CAIR has large net benefits, indicating that CAIR will likely result in improvements in overall public welfare and will provide health benefits in a highly cost-effective manner.

# G.9 References

- Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren, and P.K. Mills. 1995. "Estimated Long-Term Ambient Concentrations of PM(10) and Development of Respiratory Symptoms in a Nonsmoking Population." *Archives of Environmental Health* 50(2): 139-152.
- Agency for Healthcare Research and Quality. 2000. HCUPnet, Healthcare Cost and Utilization Project.
- American Heart Association. 2003. Heart Disease and Stroke Statistics—2003 Update. Dallas, Texas: American Heart Association.
- American Lung Association. 2002. Trends in Chronic Bronchitis and Emphysema: Morbidity and Mortality. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.
- Bala, M.V., and G.A. Zarkin. 2000. "Are QALYs an Appropriate Measure for Valuing Morbidity in Acute Diseases?" *Health Economics* 9:177-180.
- Bell, C.M., R.H. Chapman, P.W. Stone, E.A. Sandberg, and P.J. Neumann. 2001. "An Offthe-Shelf Help List: A Comprehensive Catalog of Preference Scores from Published Cost-Utility Analyses." *Medical Decision Making* 21:288-94.
- Bleichrodt, H., P.P. Wakker, and M. Johannesson. 1996. "Characterizing QALYs by Risk Neutrality." *Journal of Risk and Uncertainty* 15:107-114.

- Brock, D.W. 2002. "Chapter 14.3: Fairness and Health." In Summary Measures of Population Health: Concepts, Ethics, Measurement and Applications, C.J.L. Murray, J.A. Salomon, C.D. Mathers, and A.D. Lopez (eds.). Geneva: World Health Organization.
- Carrothers, T. J., J.S. Evans, and J.D. Graham. 2002. "The Lifesaving Benefits of Enhanced Air Quality." Harvard Center for Risk Analysis Working Paper.
- Carnethon, M.R., D. Liao, G.W. Evans, W.E. Cascio, L.E. Chambless, W.D. Rosamond, and G. Heiss. 2002. "Does the Cardiac Autonomic Response to Postural Change Predict Incident Coronary Heart Disease and Mortality? The Atherosclerosis Risk in Communities Study." *American Journal of Epidemiology* 155(1):48-56.
- Centers for Disease Control and Prevention (CDC) Health, United States. 2003. Table 30. Years of Potential Life Lost before Age 75 for Selected Causes of Death, According to Sex, Race, and Hispanic Origin: United States, Selected Years 1980–2000.
- Centers for Disease Control and Prevention (CDC). 2002. *National Vital Statistics Reports* 51(3).
- Cohen, D.J., J.A. Breall, and K.K. Ho. 1994. "Evaluating the Potential Cost-Effectiveness of Stenting as a Treatment for Symptomatic Single-Vessel Coronary Disease: Use of a Decision-Analytic Model." *Circulation* 89(4):1859-1874.
- Cohen, J.T., J.K. Hammitt, and J.I. Levy. 2003. "Fuels for Urban Transit Buses: A Costeffectiveness Analysis." *Environmental Science and Technology* 37:1477-1484.
- Cowie, M.R., A. Mosterd, D.A. Wood, et al. 1997. "The Epidemiology of Heart Failure." *European Heart Journal* 18:208-25.
- Coyle, D., D. Stieb, R.T. Burnett, P. DeCivita, D. Krewski, Y. Chen, and M.J. Thun. 2003."Impact of Particulate Air Pollution on Quality Adjusted Life Expectancy in Canada." *Journal of Toxicology and Environmental Health, Part A* 66:1847-1863.
- Cropper, M.L., and A.J. Krupnick. 1990. "The Social Costs of Chronic Heart and Lung Disease." Discussion Paper QE 89-16-REV. Washington, DC: Resources for the Future.
- de Hollander, A.E.M., J.M. Melse, E. Lebret, and P.G.N. Kramers. 1999. "An Aggregate Public Health Indicator to Represent the Impact of Multiple Environmental Exposures." *Epidemiology* 10:606-617.

- Dekker, J.M., R.S. Crow, A.R. Folsom, P.J. Hannan, D. Liao, C.A. Swenne, and E.G. Schouten. 2000. "Low Heart Rate Variability in a 2-Minute Rhythm Strip Predicts Risk of Coronary Heart Disease and Mortality From Several Causes: The ARIC Study." *Circulation 2000* 102:1239-1244.
- EPA-SAB-COUNCIL-ADV-04-002. March 2004. Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis—Benefits and Costs of the Clean Air Act, 1990-2020: Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis.
- Freeman, A.M., J.K. Hammitt, and P. De Civita. 2002. "On Quality Adjusted Life Years (QALYs) and Environmental/Consumer Safety Valuation." *AERE Newsletter* 22(1):7-12.
- Ganz, D.A., K.M. Kuntz, G.A. Jacobson, and J. Avorn. 2000. "Cost-Effectiveness of 3-Hydroxy-3-Methylglutaryl Coenzyme A Reductase Inhibitor Therapy in Older Patients with Myocardial Infarction." *Annals of Internal Medicine* 132:780-787.
- Garber, A.M., and C.E. Phelps. 1997. "Economic Foundations of Cost-Effectiveness Analysis." *Journal of Health Economics* 16:1-31.
- Gentile, J.E. 1998. *Random Number Generation and Monte Carlo Methods*. New York, NY: Springer Verlag.
- Gold, M.R., J.E. Siegel, L.B. Russell, and M.C. Weinstein. 1996. *Cost-effectiveness in Health and Medicine*. New York: Oxford University Press.
- Gold, D.R., A. Litonjua, J. Schwartz, E. Lovett, A. Larson, B. Nearing, G. Allen, M. Verrier, R. Cherry, and R. Verrier. 2000. "Ambient Pollution and Heart Rate Variability." *Circulation* 101(11):1267-1273.
- Gold, M.R., D. Stevenson, and D.G. Fryback. 2002. "HALYS and QALYS and DALYS, Oh My: Similarities and Differences in Summary Measures of Population Health." *Annual Review of Public Health* 23:115–34.

Hammitt, J.K. 2002. "QALYs versus WTP." Risk Analysis 22(5):985-1001.

Harvard Center for Risk Analysis, Catalog of Preference Scores. Available at: http://www.hcra.harvard.edu/pdf/preferencescores.pdf.

- Horsman, J., W. Furlong, D. Feeny, and G. Torrance. 2003. "The Health Utilities Index (HUI): Concepts, Measurement Properties, and Applications." *Health and Quality of Life Outcomes* 1:54.
- Hubbell, B. 2004a. "Implementing QALYs in the Analysis of Air Pollution Regulations." *Environmental and Resource Economics* (forthcoming).
- Hubbell, B. 2004b. "Health Based Cost Effectiveness of Ambient PM2.5 Reductions" Unpublished working paper.
- Johnson, F.R., and K. Lievense. 2000. Stated-Preference Indirect Utility and Quality-Adjusted Life Years. Report prepared for Health Canada, July.
- Kerridge, R.K., P.P. Glasziou, and K.M. Hillman. 1995. "The Use of "Quality-Adjusted Life Years" (QALYs) to Evaluate Treatment in Intensive Care." *Anaesthia and Intensive Care* 23:322-31.
- Kind, P. 1996. The EuroQoL Instrument: An Index of Health-Related Quality of Life. Quality of Life and Pharmacoeconomics in Clinical Trials, Second Edition, B. Spilker (ed.), pp. 191-201. Philadelphia, PA: Lippincott-Raven Publishers.
- Krewski, D., R.T. Burnett, M.S. Goldbert, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz, and W.H. White. 2000. *Reanalysis of the Harvard Six Cities Study* and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute, Cambridge, MA.
- Kuntz, K.M, J. Tsevant, L. Goldman, and M.C. Weinstein. 1996. "Cost-Effectiveness of Routine Coronary Angiography after Acute Myocardial Infarction." *Circulation* 94(5):957-65.
- Kunzli, N., R. Kaiser, S. Medina, M. Studnicka, O. Chanel, P. Filliger, M. Herry, F. Horak Jr., V. Puybonnieux-Texier, P. Quenel, J. Schneider, R. Seethaler, J-C Vergnaud, and H. Sommer. 2000. "Public-Health Impact of Outdoor and Traffic-Related Air Pollution: A European Assessment." *The Lancet* 356:795-801.
- Kunzli, N., S. Medina, R. Kaiser, P. Quenel, F. Horak Jr, and M. Studnicka. 2001.
  "Assessment of Deaths Attributable to Air Pollution: Should We Use Risk Estimates Based on Time Series or on Cohort Studies?" *American Journal of Epidemiology* 153(11):1050-55.

- Liao, D., J. Cai, W.D. Rosamond, R.W. Barnes, R.G. Hutchinson, E.A. Whitsel, P.
  Rautaharju, and G. Heiss. 1997. "Cardiac Autonomic Function and Incident Coronary Heart Disease: A Population-Based Case-Cohort Study. The ARIC Study. Atherosclerosis Risk in Communities Study." *American Journal of Epidemiology* 145(8):696-706.
- Liao, D., J. Creason, C. Shy, R. Williams, R. Watts, and R. Zweidinger. 1999. "Daily Variation of Particulate Air Pollution and Poor Cardiac Autonomic Control in the Elderly." *Environmental Health Perspectives* 107:521-5.
- Magari, S.R., R. Hauser, J. Schwartz, P.L. Williams, T.J. Smith, and D.C. Christiani. 2001.
  "Association of Heart Rate Variability with Occupational and Environmental Exposure to Particulate Air Pollution." *Circulation* 104(9):986-91.
- Miller, B.G., and J.F. Hurley. 2003. "Life Table Methods for Quantitative Impact Assessments in Chronic Mortality." *Journal of Epidemiology and Community Health* 57:200-206.
- Moolgavkar, S.H. 2000. "Air Pollution and Hospital Admissions for Diseases of the Circulatory System in Three U.S. Metropolitan Areas." *Journal of the Air Waste Management Association* 50:1199-206.
- Murray, C.J.L., J.A. Salomon, C.D. Mathers, and A.D. Lopez. 2002. "Summary Measures of Population Health: Concepts, Ethics, Measurement and Applications." Geneva: World Health Organization.
- National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.
- Neumann, P. 2003. "A Web-based Registry of Cost-utility Analyses." *Risk in Perspective* 11(3).
- Peters, A., D.W. Dockery, J.E. Muller, and M.A. Mittleman. 2001. "Increased Particulate Air Pollution and the Triggering of Myocardial Infarction." *Circulation* 103:2810-2815.
- Pliskin, J.S., D.S. Shepard, and M.C. Weinstein. 1980. "Utility Functions for Life Years and Health Status." *Operations Research* 28:206-224.

- Pliskin, J.S., W.B. Stason, and M.C. Weinstein. 1981. "Coronary Artery Bypass Graft Surgery: Clinical Decision Making and Cost-Effective Analysis." *Medical Decision Making* 1(1):10-28.
- Poloniecki, J.D., R.W. Atkinson, A.P. de Leon, and H.R. Anderson. 1997. "Daily Time Series for Cardiovascular Hospital Admissions and Previous Day's Air Pollution in London, UK." *Occupational and Environmental Medicine* 54(8):535-40.
- Pope, D.A., R.T. Burnett, M.J. Thun, E.E. Cale, D. Krewski, K. Ito, and G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141.
- Rabl, A. 2003. "Interpretation of Air Pollution Mortality: Number of Deaths or Years of Life Lost?" *Journal of the Air and Waste Management Association* 53:41-50.
- Russell, M.W., D.M. Huse, S. Drowns, E.C. Hamel, and S.C. Hartz. 1998. "Direct Medical Costs of Coronary Artery Disease in the United States." *American Journal of Cardiology* 81(9):1110-5.
- Salkeld, G., P. Phongsavan, and B. Oldenburg. 1997. "The Cost-Effectiveness of a Cardiovascular Risk Reduction Program in General Practice." *Health Policy* 41:105-19.
- Salomon, J.A., and C.J.L. Murray. 2003. "A Multi-Method Approach to Measuring Health-State Valuations." *Health Economics*, online.
- Samet, J.M., S.L. Zeger, F. Dominici, F. Curriero, I. Coursac, D.W. Dockery, J. Schwartz, and A. Zanobetti. June 2000. *The National Morbidity, Mortality and Air Pollution Study: Part II: Morbidity, Mortality and Air Pollution in the United States.* Research Report No. 94, Part II. Cambridge, MA: Health Effects Institute.
- Schwartz, J. 1993. "Particulate Air Pollution and Chronic Respiratory Disease." *Environmental Research* 62:7-13.
- Singer, P., J. McKie, H. Kuhse, J. Richardson, and J. Harris. 1995. "Double Jeopardy and the Use of QALYs in Health Care Allocation." *Journal of Medical Ethics* 21:144-157.

- Smith, K.J., and R.R. Pesce. 1994. "Pulmonary Artery Catheterization in Exacerbations of COPD Requiring Mechanical Ventilation: A Cost-Effectiveness Analysis." *Respiratory Care* 39:961-7.
- Stinnett, A.A., M.A. Mittleman, M.C. Weinstein, K.M. Kuntz, D.J. Cohen, L.W. Williams, P.A. Goldman, D.O. Staiger, M.G.M. Hunink, J. Tsevat, A.N.A. Tosteson, and L. Goldman. 1996. "The Cost-effectiveness of Dietary and Pharmacologic Therapy for Cholesterol Reduction in Adults." In *Cost-effectiveness in Health and Medicine*, Gold, M.E., J. E. Siegel, L.B. Russell, and M.C. Weinstein (eds.). New York: Oxford University Press.
- Stouthard, M.E.A., J.L. Essink-Bot, G.J. Bonsel, J.J. Barendregt, P.G.N. Kramers, H.P.A. Van de Water, and L.J. Gunning-Schepers. 1997. "Disability Weights for Diseases in the Netherlands." Rotterdam: Department of Public Health, Erasmus University Rotterdam.
- Suwa, T. J.C. Hogg, K.B. Quinlan, A. Ohgami, R. Vincent, and S.F. van Eeden. 2002. "Particulate Air Pollution Induces Progression of Atherosclerosis." *Journal of the American College of Cardiology* 39:935-942.
- Teng, T.O., M.E. Adams, J.S. Pliskin, D.G. Safran, J.E. Siegel, M.C. Weinstein, and J.D. Graham. 1995. "Five Hundred Life-Saving Interventions and Their Cost-Effectiveness." *Risk Analysis* 15:369-390.
- Tsuji, H., M.G. Larson, F.J. Venditti, Jr., E.S. Manders, J.C. Evans, C.L. Feldman, and D. Levy. 1996. "Impact of Reduced Heart Rate Variability on Risk for Cardiac Events. The Framingham Heart Study." *Circulation* 94(11):2850-2855.
- U.S. Environmental Protection Agency (EPA). 1996. Review of the National Ambient Air Quality Standards for Particulate Matter: Assessment of Scientific and Technical Information. Report no. EPA/4521R-96-013. Research Triangle Park, NC: Office of Air Quality Planning and Standards.
- U.S. Environmental Protection Agency (EPA). 2000. *Guidelines for Preparing Economic Analyses*. EPA 240-R-00-003.
- U.S. Environmental Protection Agency (EPA). 2004. Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines.

- U.S. Food and Drug Administration (FDA). 2004. Bar Code Label Requirements for Human Drug Products and Biological Products; Final Rule. Federal Register: February 26, 2004 Volume 69 (38): 9119-9171.
- U.S. Office of Management and Budget (OMB). 2003. Circular A-4: Regulatory Analysis, September 17, 2003. Available at: http://www.whitehouse.gov/omb/ circulars/a004/a-4.pdf.
- Vos, T. 1999a. Final worksheet VM1 COPD.xls, prepared for Victoria Burden of Disease Study. Available at: http://www.dhs.vic.gov.au/phd/bod/daly.htm.
- Vos, T. 1999b. Final worksheet VL2 IHD.xls, prepared for Victoria Burden of Disease Study. Available at: http://www.dhs.vic.gov.au/phd/bod/daly.htm.
- Wittels, E.H., J.W. Hay, and A.M. Gotto, Jr. 1990. "Medical Costs of Coronary Artery Disease in the United States." *American Journal of Cardiology* 65(7):432-40.

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